THE JOURNAL OF PERIODONTOLOGY

July
1957

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 28
NUMBER 3

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The Journal of Periodontology
is dedicated to

Dr. Gillette Hayden

and

Dr. Grace Rogers Spalding

for their pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

THE JOURNAL OF PERIODONTOLOGY
(J. Periodont.)

Published Quarterly—January, April, July, and October—by

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Publication Office: 2620 FLETCHER ST., CHICAGO 18, ILL.
Editorial Office: 1121 WEST MICHIGAN ST., INDIANAPOLIS 2, INDIANA
Single copies of this number may be obtained from
Editorial Office at $2.00 each
Subscription Price $8.00
All foreign subscriptions to be paid in United States money

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The Journal of Periodontology, Vol. 28, No. 3, July, 1957. Published by the American Academy of Periodontology. Publication office, 2620 Fletcher St., Chicago 18, Ill. Editorial office, 1121 W. Michigan St., Indianapolis 2, Ind. Yearly subscription, $8.00. Single copy, $2.00. Entered as second-class matter at the post office at Chicago, Ill. Published quarterly. Copyright 1957 by the American Academy of Periodontology.

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

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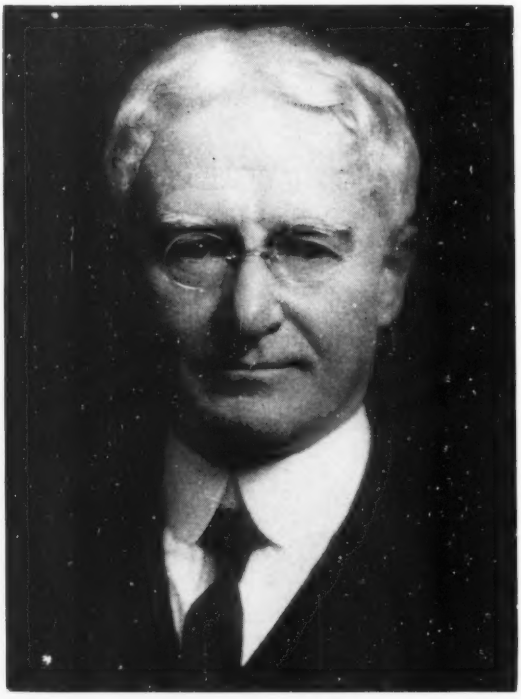

DR. WILLIAM JOHN GIES

1872 - 1956

To write a summary of Dr. William J. Gies' activities, or to write a eulogy of him would take far more space than is available in this issue of THE JOURNAL OF PERIODONTOLOGY. At this time it must suffice to record the deep appreciation of the members of the Academy of Periodontology, and of the Editor of THE JOURNAL OF PERIODONTOLOGY, for the inspiration, the guidance, the help and the contributions of Dr. William J. Gies. Though he was not a dentist his influence for good in dentistry has been equalled by few. It is a privilege, to dedicate to him this issue of the Journal.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY-SEVEN

Extension of Exudate Into Supporting Structures of Teeth in Marginal Periodontitis

by HENRY M. GOLDMAN, D.M.D.,* *Boston, Mass.*

INFLAMMATION is the local reaction in tissues following injury or irritation; the agents causing the injury may be of bacteriologic, physical, chemical, or traumatic nature. This reaction, inflammation, is accompanied or followed by reparative or regenerative healing. Chronic inflammation is a process in which the exudative changes are prolonged and proliferation of connective tissue, a fundamental part of the response of the body to injury, tending to repair the damage, is a prominent feature.

In the case of periodontal disease, the causative agents are chronic and low-grade in nature and as a result the inflammatory response is so characterized. The predominant cells are lymphocytes, plasma cells, and macrophages, although in the subsulcular area where the injury is more severe, polymorphnuclear leukocytes may be present. The proliferative activity leading to the production of connective tissue varies and, hence, in some cases, in the late stages of the disease process, the gingival color assumes various hues from off-coral-pink to bluish-red.

In the early stages of gingival disease, the inflammation is localized to the subsulcular area. As the disease progresses, this inflammatory response extends throughout the gingiva and subsequently is seen in the subjacent structures. An understanding of the pathway of the inflammatory process in marginal periodontitis is of great importance, for associated with it is the resorption of the alveolar housing of the tooth. This problem has been discussed by various investigators, the most comprehensive report being by Weinmann (1941). He reviewed the literature and described in detail the pathway as progressing from the gingiva into the bone marrow spaces following the course of the blood vessels. In relatively few instances did the inflammation penetrate into the periodontal membrane. He believed that the distribution of the blood vessels explained the course followed by the infiltrate.

Investigation into this problem was undertaken in order to obtain a three-dimensional picture of the pathway of the inflammatory infiltrate into the subjacent tissues. Mesiodistal as well as buccolingual sections were studied.

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Support for this investigation was derived from funds obtained from the Bristol-Myers Company.

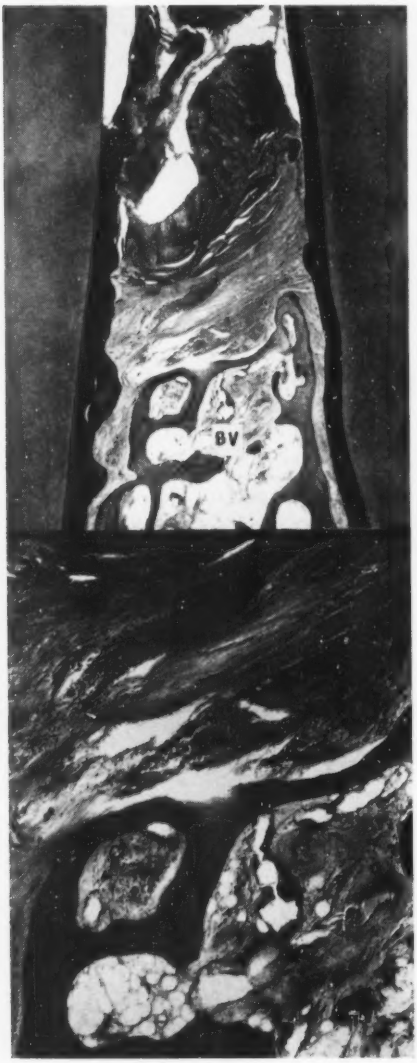

Fig. 1

MATERIAL AND METHODS

Mesiodistal and buccolingual sections from fifteen human jaws affected by periodontal disease prepared for microscopic study and stained with hematoxylin and eosin were studied. The sections were those through the center of the teeth in both buccolingual and mesiodistal directions; sections through the interdental area, cut buccolingually, were also investigated.

Fig. 2

FINDINGS

The pathway of inflammatory infiltrate from the gingiva into the subjacent structures seemingly follows the course of the vascular supply (lymph drainage). In the three dimensional view the findings can be best described by selected photomicrographs illustrating the pertinent data.

Figure 1 contains both low- and high-power photomicrographs of a mesiodistal section between two mandibular premolars. The dense inflammatory infiltrate in the gingiva and the accumulations between the transseptal fiber groups may be noted in the low-power photomicrograph. Calculus is adherent to the tooth surface and covers the gingiva. The exudate is especially dense subjacent to the covering epithelium. In the lower photomicrograph the infiltrate is located not only in between the dense collagen groups of the transseptal fibers but also in the marrow spaces of the bone.

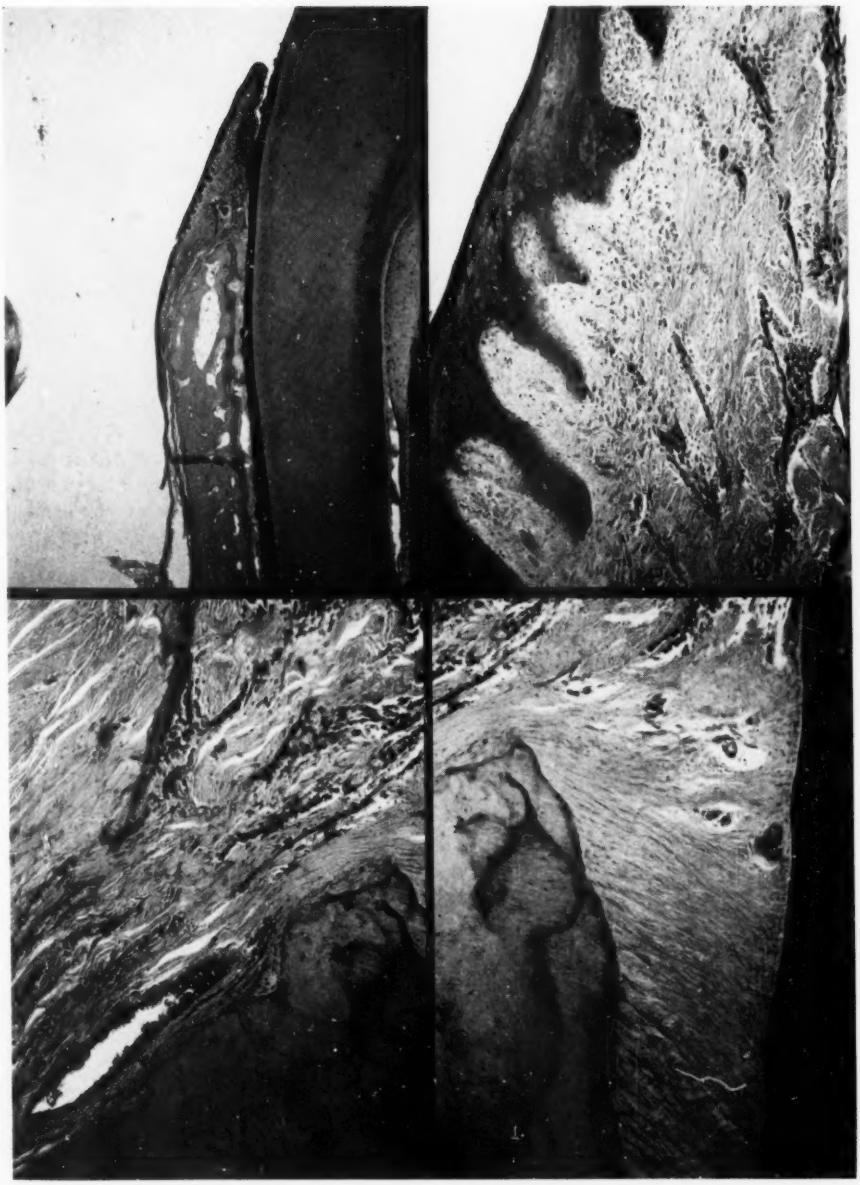

Fig. 3

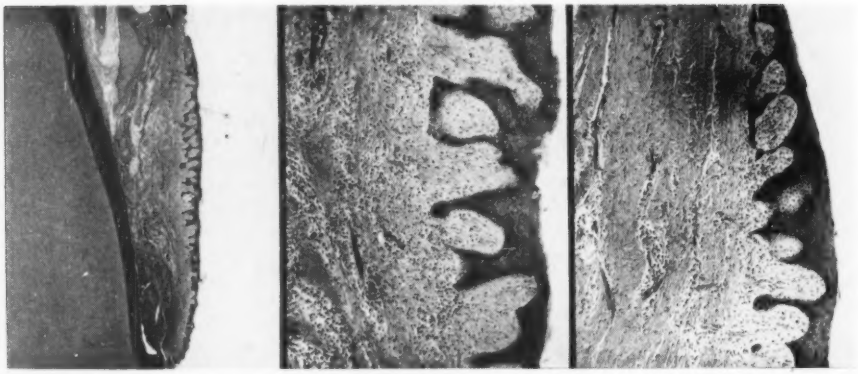

Fig. 4

It is interesting to note that the periodontal membrane on both sides is free from any exudate (low-power). The intra-alveolar artery may be seen at "BV."

Figure 2 also shows a mesiodistal section of two teeth, the structures reading from left to right: dentin, cementum, periodontal membrane, alveolar process, periodontal membrane, cementum, and dentin. In the superior portion of the photomicrograph, one sees the transseptal fibers above the alveolar crest and the gingival corium directly above. This latter tissue contains a dense, inflammatory infiltrate; inflammatory cells are seen between the transseptal fibers. A densely packed exudate is seen at the crestal region and below in between the two sections of bone. With no bone between it and the tooth, the infiltrate can be seen extending into the periodontal membrane. On the other side, however, no exudate in the periodontal membrane is evident. Note that the marrow spaces of the bone are fibrosed.

Figure 3 is a buccolingual section through the tooth directly in the pulp chamber (central section). In the upper left photomicrograph the following structures may be seen, reading from left to right: buccal gingivae, buccal alveolus, periodontal membrane, cementum, dentin, and pulp. There is a dense, inflammatory infiltrate adjacent to the sulcular epithelium. The group of gingival fibers arising in the cementum above the alveolar crest and extending into the gingiva travelling apically on the outer aspect of the alveolus, may also be noted. In the lower left photomicrograph, a high-power of the section described above, a large blood vessel above the crest is seen as well as the exudate in streak-like fashion in between the bundles of connective tissue. In the photomicrograph in the upper right, the infiltrate is seen in the gingival corium away from the subsulcular area. Of great interest is the lower right photomicrograph illustrating the periodontal membrane in the crestal region; no infiltrate is present.

Thus, it is evident in this section that the infiltrate is densest at the site of the wound area, the subsulcular area, and then extends through the gingival corium travelling into the attached gingiva. The periodontal membrane is spared.

Figure 4 represents a similar situation as seen in the previous illustration except that the exudate is far more heavy in the gingival corium and is seen in the papillary layer of the gingiva. In the low-power photomicrograph, one can observe, nevertheless, that the periodontal membrane contains no inflammatory cells.

Figure 5 (upper left) is a buccolingual section showing the buccal gingiva and the alveolar process. The gingiva contains

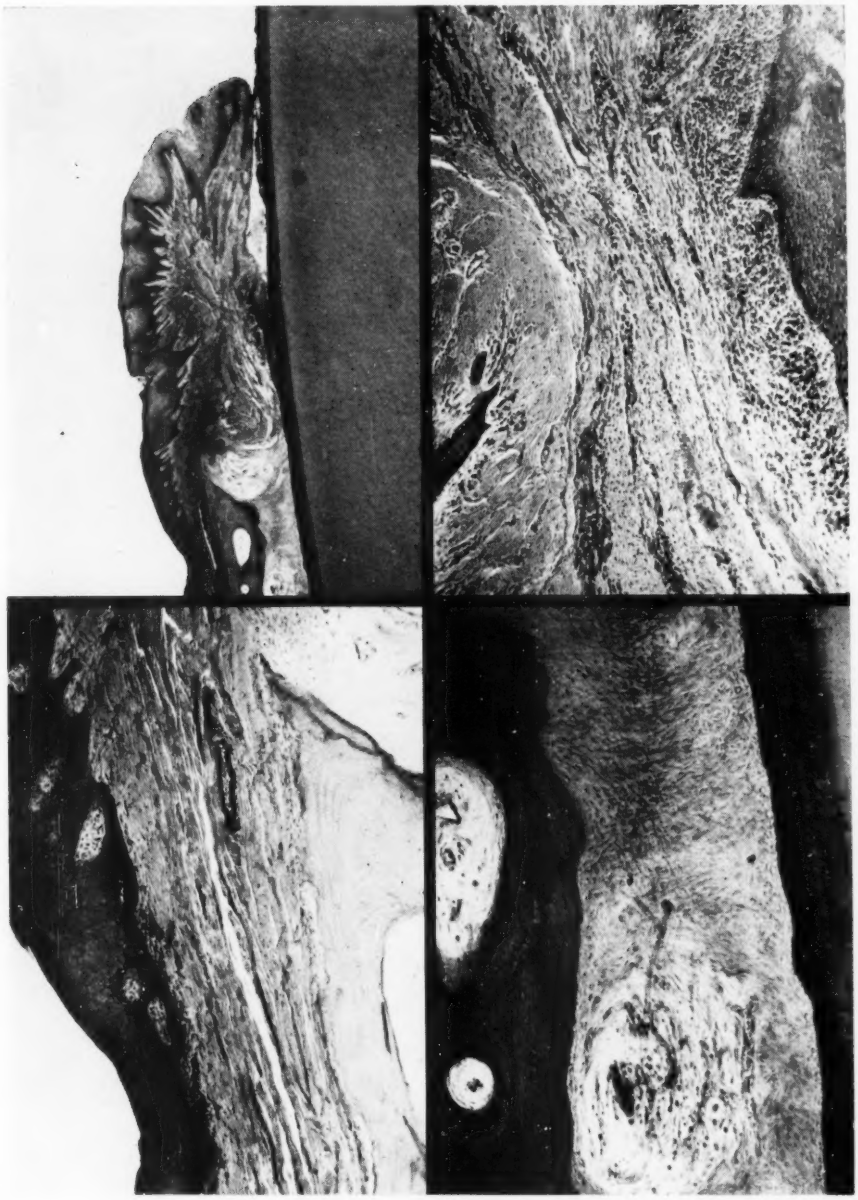

Fig. 5

numerous inflammatory cells diffusely spread throughout and extending into the gingival tissue covering the alveolar process. In this case, however, as seen in the lower right photomicrograph, there is an accumulation of inflammatory cells in be-

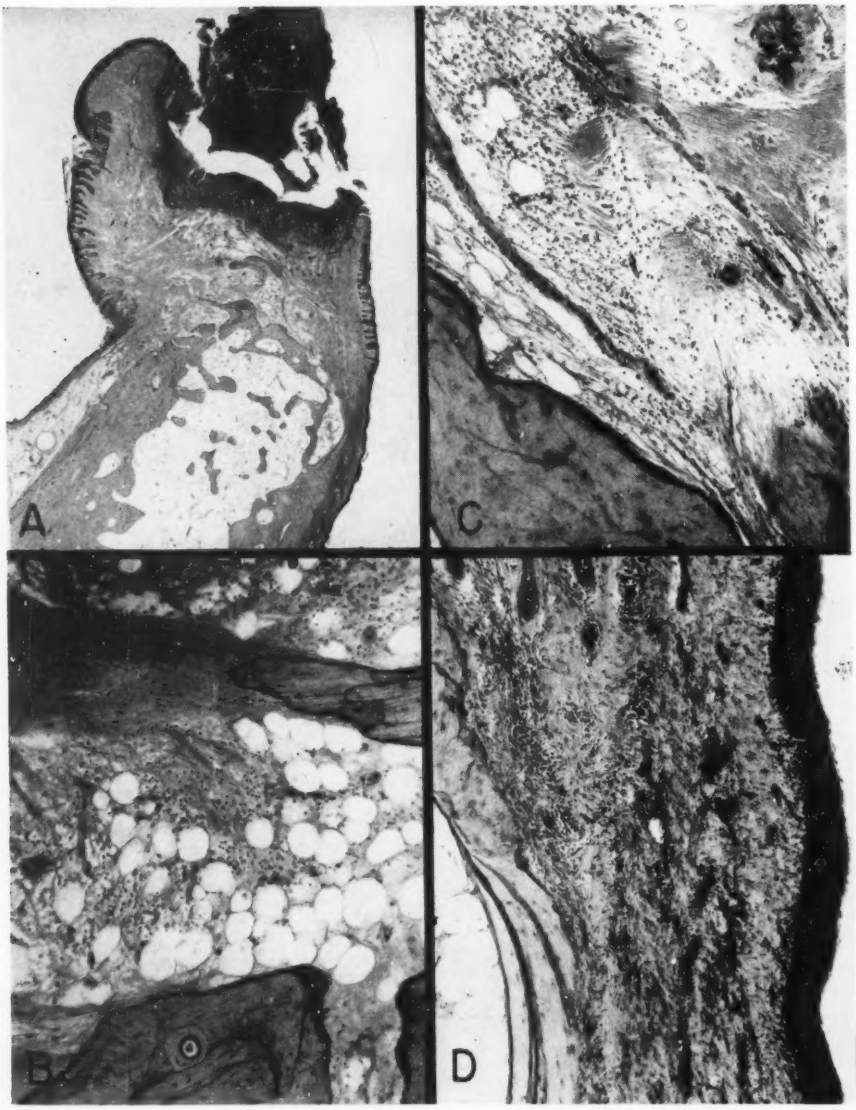

Fig. 6

tween the fiber groups of the periodontal membrane. While the infiltrate is not densely packed, it represents an active infiltrate into this area. Thus, in contrast to the two previous illustrations, in this case, an infiltrate is seen in the periodontal membrane.

Figure 6 is a buccolingual section of an interdental area in an advanced marginal periodontitis; clinically an interdental crater was present. The inflammatory infiltrate is especially heavy at the base of the gingival crater. The alveolar process has been greatly reduced and one notes a

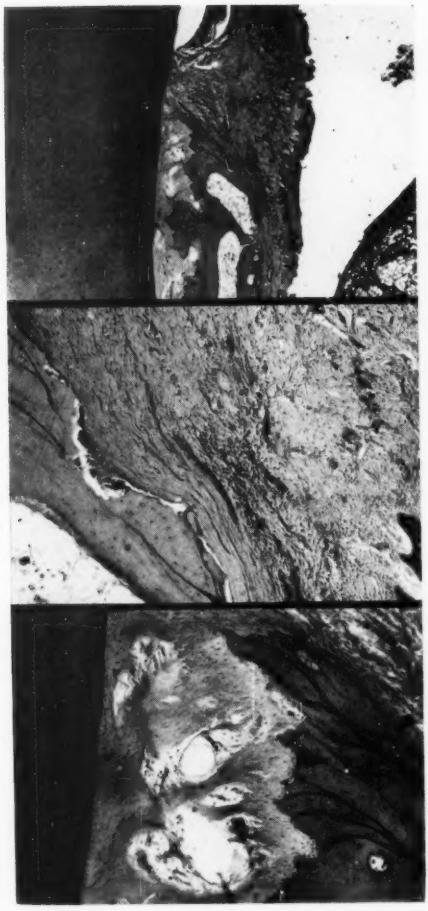

Fig. 7

bone trabecula lying horizontally, subjacent to the crater area. In higher power (B) the exudate is seen in the marrow space between the bone trabeculae. Thus, the pathway consists of apical penetration into the alveolar process. However, the inflammatory infiltrate also follows a course in the gingival wall itself (C) on the outer surface of the process, and in "D," a dense infiltrate may be noted situated in the alveolar mucosa.

Figure 7 (low power) represents a buccolingual section showing the lingual gingiva and alveolar process. The dense inflammatory infiltrate in the gingiva is evident and its extension may be seen in the middle illustration. Here the inflammatory cells are seen in between the collagen fibers. An area of active bone resorption on the periosteal side of the alveolus may be noted. The great interest in this case, however, centers in the lower photomicrograph where one sees resorption of the alveolar bone associated with occlusal traumatism. Despite this fact no inflammatory cells are seen. Thus, the extension of the exudate has taken place as in other instances of gingival disease and the two processes have remained distinct.

Figure 8 are low- and high-power photomicrographs of a buccolingual section of the distal root of a mandibular first molar. The occlusal wear of the tooth may be seen. High-power examination of this section reveals that there is a horizontal lesion consistent with occlusal trauma on the lingual aspect, while on the buccal, the evidence of tension in the attachment apparatus is present. The high-power photomicrograph represents the crestal portion of the buccal alveolus and a distinct osteoblastic layer adjacent to the periodontal membrane is present. This constitutes an active production of bone usually seen on the tension side in the occlusal traumatic lesion. Despite the fact that there is a marked gingival inflammation there is no evidence of any inflammatory exudate in the periodontal membrane.

DISCUSSION

Gingival inflammation is first seen in the subsulcular area. Here, the inflammatory cells become densely packed as the disease process progresses. The inflammatory process does not become confined to this area but soon extends into the deeper structures. The gingival corium as a whole soon contains either sparsely distributed groups of inflammatory cells or in some instances occupies the entire corium after destruction of the collagen element.

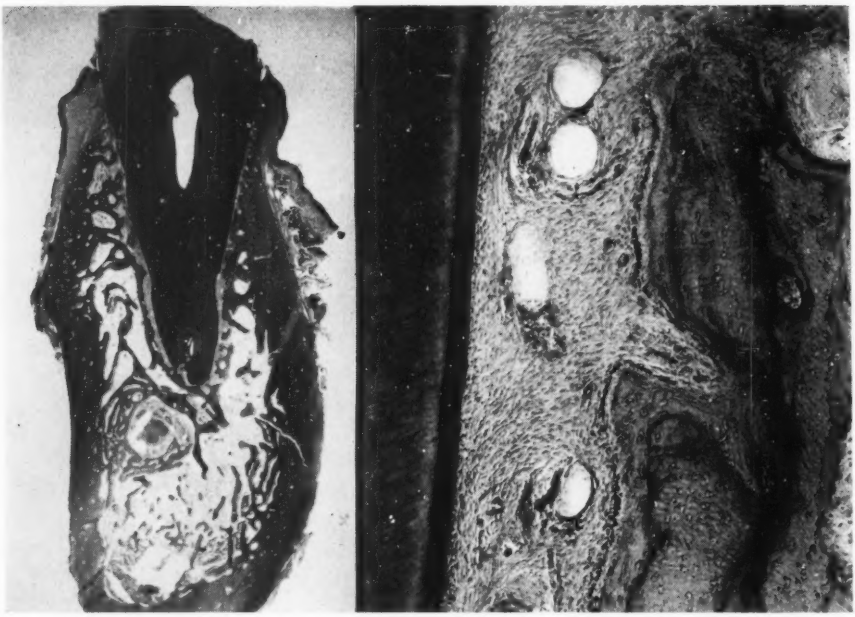

Fig. 8

Progressing from the gingiva (marginal area—buccal or lingual) the inflammatory exudate extends throughout the attached gingiva, at times into the alveolar mucosa. Thus the inflammatory cells are seen outside the alveolar process. Occasionally, scattered inflammatory cells are found in the marrow spaces of the crestal region of the buccal and lingual alveolar processes and in the periodontal membrane (Fig. 5).

The infiltrate in the interdental papilla progresses apically through the transseptal fibers and into the marrow spaces of the interdental septum. However, the progress of inflammation of this area does not solely extend into the bone but also follows the pattern seen in the marginal area. Buccolingual sections of the interdental area discloses that the pathway extends not only into the bone but also outside, buccally or lingually, of the bone in the gingiva itself (Fig. 6). Occasionally, an infiltrate is found in the periodontal membrane adjacent to the interdental septum (Fig. 2).

The inference of these microscopic findings are that the changes in color, consistency, form; etc., seen in gingival disease are caused by the apical progression of the inflammatory elements in the gingival tissue (attached gingiva-alveolar mucosa). At times in severe gingival disease the attached gingiva-alveolar mucosa demarcation line becomes obliterated and the change in color extends from the gingival margin down almost to the mucobuccal fold. This is the result of the extension of a dense or severe inflammatory process. Inflammation in the interdental papilla causes changes in this region and sometimes almost is limited thereto. As the disease process advances, the spread occurs to subjacent structures. This is an expression of the apical extension of inflammation in the gingiva.

For the most part the periodontal membrane is not affected by the extension of the gingival inflammation; however, in some cases focal accumulations of inflam-

matory cells are found near the vascular structures in between groups of the collagen fibers. Seemingly there is no direct extension from the gingival area but an entrance is gained laterally through the openings from adjacent marrow spaces. This was found not only in the mesiodistal but also the buccolingual sections. Progression of exudate into the periodontal membrane area is seen in most instances of infrabony pockets especially if the orifice is broad, or if two walls, proximal and buccal or lingual, are present.

The changes which occur when the periodontium is subjected to both marginal injury and occlusal traumatism consist of the two processes going on simultaneously. Recently, Macapanpan and Weinmann in a study on the influence of injury to the periodontal membrane on the spread of gingival inflammation, concluded that if the periodontal membrane is injured by tension, the inflammation might spread indirectly into it, while if the periodontal membrane is normal, the inflammation will spread into the bone marrow. Injury from tension alone does not lead to leukocytic infiltration. They felt that these findings were of interest in that in severe occlusal injuries to the periodontium in the presence of gingival inflammation, there may be a spread of the inflammation into the periodontal membrane which would not heal unless both the occlusal and gingival causations were eliminated. Study of necropsy material, however, does not confirm the above experimental findings in that no inflammatory cells were seen in the periodontal membrane on the tension side. It may be that an *acute, continuously active, and severe* gingival irritation is necessary (as in the above experiment) whereas in clinical periodontal manifestations, for the most part the gingival condition is chronic in nature.

SUMMARY

The course of the inflammatory infiltrate in marginal periodontitis consists of apical progression into the alveolar processes (buccal, interdental, and lingual) and into the gingival tissue itself. Occasionally focal accumulations of inflammatory cells are found in the crestal region of the periodontal membrane. The changes which occur when the periodontium is subjected to both marginal injury and occlusal traumatism are independent.

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Weinmann, J. P.: Progress of Gingival Inflammation into the Supporting Structures of the Teeth. J. Periodont. 12:71, 1941.

TUFT'S BERKSHIRE CONFERENCE HONORS DEAN MAYNARD K. HINE

Tuft's University School of Dental Medicine held its Eighth Annual Conference on Periodontology and Oral Pathology in mid-June at Eastover in Lenox, Massachusetts. The conference, which was well attended, proved to be stimulating, instructive, and relaxing for all who attended. The program was planned by Dr. Irving Glickman and included many scientific discussions, a morning with Dr. Joseph Bernier and microscopes for all, as well as afternoons free to enjoy the beautiful Berkshire Hills.

At the opening dinner, Dean C. D. Marshall-Day awarded Dr. Maynard K. Hine, Editor of THE JOURNAL OF PERIODONTOLOGY the "1957 Award for Leadership in Periodontology." This award is given annually to some one who in the opinion of the Periodontology Department at Tuft's has made noteworthy contributions to the science of Periodontology.

Bone Implants in Periodontal Diseases—A Further Study*

by W. G. Cross, M.B., M.S., B.D.S.,** *London, England*

RECENTLY two reports[1, 2] have been published by the author concerning bone grafts by way of a preliminary study, and it is the purpose of this article to take the study somewhat further. Since that time, articles have been published by Forsberg[3] and Krømer,[4] and reference will be made to these later.

Literature Review

Beube and Silvers[5] reported as long ago as 1934, on the insertion of boiled bovine bone powder into surgically produced cavities in jaws of animals, with generation of new bone. They subsequently reported on the use of this type of bone in humans in 14 cases—of these 5 had periodontal disease, one being successful.[6] The four failures in this group were considered to be due to lack of closure of the pockets, with consequent suppuration. They go on to claim that the use of bone chips, or the insertion of a single piece of bone, would not be so successful. No local or general reaction was noted.

In a further report, Beube[7] noted bone regeneration in five patients treated by implantation of boiled bovine bone powder, where previous conservative treatment had not produced any regeneration. A detailed description of the preparation of bovine bone, its indications and mode of use, is given by Beube in his textbook.[8] The indications given are for infrabony pockets of 7 mm. or more, around single-rooted teeth, where the pockets have not been closed by curettage. Radiographs of 5 cases are shown. The technique consists, in essence, of the raising of a flap (the incision being kept at least 4 mm. from the gingival margin,) the removal of granulation tissue, bone trimming if indicated, removal of all cementum, and insertion of a paste of bone powder, fibrin foam and thrombin (or powdered beef plasma, as suggested by Fox). The wound is sutured by very close interrupted sutures, which are removed in 3-5 days. A bland mouthwash is prescribed. No mention is made of antibiotic cover or post-operative antibiotics, splinting if teeth are loose, or post-operative dressings. The results as assessed by the radiographs of cases shown are good, in that filling in of resorbed bone has occurred. Pre-operative and post-operative radiopaque points were not used (except for the use of gutta-percha points in two cases pre-operatively) and thus no indication of pocket depth was given.

Forsberg[3] described the use of os purum in the treatment of 11 cases. Os purum, formerly prepared by the Swedish firm Pharmacia, but now no longer in production, was ox bone treated physicochemically to remove fat, connective tissue and protein. It was prepared in various forms including granules of varying degrees of fineness, and was used in this form by Forsberg. Following preliminary examination and treatment to remove any aetiological factors found, a flap was raised and all granulation tissue removed. Ground os purum was then introduced and packed as tightly as possible up to just beyond the alveolar margin. This was followed by careful and accurate suturing and the application of a previously prepared transparent fixation splint, which permitted inspection of the dressing. This author reported that he has recently modified the technique by using autogenous bone taken from the patient's iliac crest, in the form of chips. These chips were then ground with a mortar and pestle, and immediately used in the pockets. A table was given showing that one case was, after five months, classified as having an 'excellent' result, seven cases after 2-12 months, as 'satisfactory' and three as 'not satisfactory.' No radiographs were shown and the proportion of cases treated by os purum to those with autogenous chips was not stated. The longest observation period was one year, the shortest, two months. The author did not mention the use of antibiotics at any stage, or whether the cementum was removed or in fact curetted at all.

Two articles have been published by Krømer. In the second[4] he stated that he had treated 13 cases of intrabony pockets with deep frozen stored bone, kept in 1:1000 merthiolate solution which was changed once a fortnight. He advocated a flap which combined a vertical and horizontal incision, and left the interdental papilla overlying the intrabony pocket intact. Thorough curettage was carried out to remove granulations, epithelial rests, calculus and necrotic cementum. The area was washed out with physiological saline and small chips of spongiosa packed in. Thrombin was pipetted into the area, and the flap sutured. Surgical cement was placed in the interproximal space, and sutures were removed after 5-7 days.

Preliminary treatment included removal of calculus, and grinding, nine days pre-operatively; the day before operation an antibiotic umbrella was commenced (type not specified) and maintained for 3-5 days. No exploration of the pocket was done during the early stages.

He found that out of 13 cases, one was a failure, due to suppuration and loss of all the implant, and in three cases the exfoliation of one or two chips

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**Senior Lecturer and Head of Department of Periodontology.

was followed by healing. He warned against overpacking with chips at operation. The observation time for this series of cases was from 3-20 months. Radiographs of one treated case were shown.

In a recent article Linghorne[9] reported bone regeneration had been induced by means of autogenous grafts in artificially produced epithelialised pockets in 10 dogs. The results indicated substantial reattachment and regeneration of bone, one of this author's interesting suggestions being that the removal of epithelium, a clinically difficult task, may not be necessary.

PRESENT STUDY

Material of present study relates to 27 patients treated in the Department of Periodontology, Institute of Dental Surgery, London, between March 1952 and March 1957; the majority by the author,

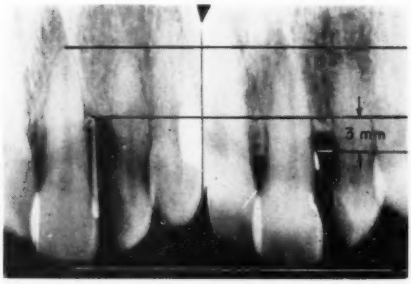

Fig. 1. Autogenous implant. Pocket before treatment 10 mm., and four years eleven months after, 2 mm.

the remainder by the staff of the Department and research associates. The total number of implants for these cases totalled 35, one patient requiring a second implantation in the same area, and six others receiving implants in more than one area. Of these patients, 11 were male and 16 female, and the age distribution was 15-20 years—2; 21-30 years—10; 31-40 years—10; 41-50 years—3; 51-60 years—2. Some form of splinting was used in 8 cases. The initial condition was an intrabony pocket varying from 6 to 16 millimeters in depth, except for two cases where an attempt was made to raise the level of a more or less flat interdental crest of alveolus (Figs. 1, 2).

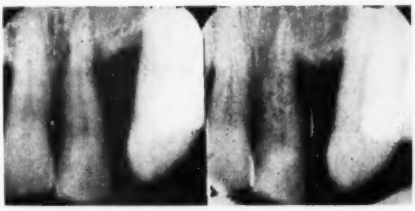

Fig. 2. Homogenous implant; /1 and adjacent alveolus subsequently examined histologically, three and one-half months later.

The teeth involved were mainly the upper or lower six anteriors, but premolars or molars were involved in six cases.

Types of Implant

Autogenous grafts were used in the first three cases of the series. Using a small trephine in the handpiece, the cortical layer was removed from an edentulous area in the patient and some cancellous bone then removed and cut into small chips for insertion. One of these cases is illustrated (Fig. 1). At about this time the author learned of a bone bank at an adjacent general hospital and the next 20 cases were treated with this material (Figs. 2-14 inclusive). This bone consisted of bone removed at some surgical procedure, cultured, placed in a sterile glass jar, and stored in a deep freeze unit. Twenty-four hours before operation it is left in a penicillin-streptomycin solution.

In the four most recent cases, bovine bone, treated with ethylene-diamine, has been used (Figs. 15, 16). The method of

Fig. 3. Homogenous implant. Pocket before treatment, 10 mm., and fourteen and one-half months after, 4 mm.

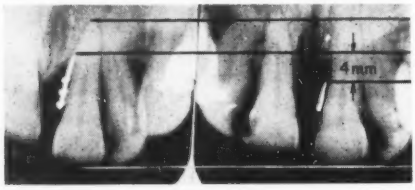

Fig. 4. Homogenous implant. Pocket before treatment, 8 mm., and 20 months after, 4 mm.

preparation of this bone and its use in animals of other species with histological results, was demonstrated on behalf of Losee at the Annual Conference of the British Dental Association, July 1956. Losee[10] states that no foreign body or immunological reactions have been observed with this material.

The advantages are that the material can be ground so as to pass through a sieve of appropriate mesh, thus ensuring a convenient particle size, can be autoclaved, and contains no organic material. It would appear to be similar to the os purum used by Forsberg.

The Problem

The ideal outcome of treatment would be the clinical elimination of the pocket, as assessed by attempting to introduce thin stainless band material (the usual pocket measuring probe is too thick) coupled with the return to a normal radiographical appearance, i.e., a complete filling in of the bony defect by bone or normal trabecular pattern and density and the reconstruction of a lamina dura, and of a periodontal space of normal even width. This is not to say that an outcome which does not measure up to these exacting criteria cannot be satisfactory to all intents and purposes.

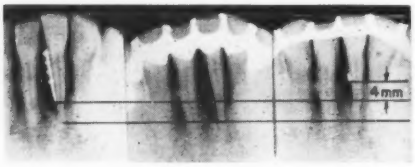

Fig. 5. Homogenous implant. Pocket before treatment, 10 mm., 6 weeks later, and then 20 months after, 4 mm.

It seems that there is a fair measure of agreement as to the procedures necessary to achieve reattachment, (McCall,[11] Schaffer and Zander,[12] Carranza,[13] Goldman,[14] Cross[15]) namely the complete removal of deposits on the root surface and granulation tissue present, and according to some of these authors, removal of some, if not all, cementum. Many reports and case histories have been published giving clinical and radiographical evidence of such reattachment, and not infrequently bone regeneration has been demonstrated. Some of these cases have demonstrated the regeneration of quite a considerable amount of bone

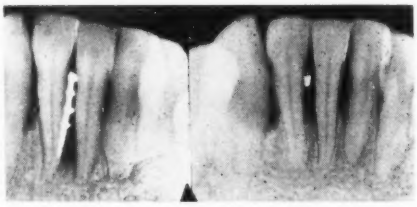

Fig. 6. Homogenous implant. Pocket before treatment, 10 mm., and twenty-three and one-half months after, 2 mm.

and this brings one to ask what the indications for the use of bone as an implant are. The exposure time of the radiograph is a factor to be considered; an under-exposure will often reveal traces of 'ghost' bone with the capacity for remineralization, such traces being obliterated with a longer exposure, and the apparent loss may not necessitate implanted bone to make it good.

Two types of intrabony pocket often respond to curettage without bone implantation: the very deep, narrow type, and the tortuous type where the pocket terminates in a bony 'cavern' on some other aspect of the root surface, the area of resorption being covered coronally with normal alveolus.

There are many other intrabony pockets however, with wide orifices, e.g. the type

in which there is, especially interdentally, a semilunar outline of bone loss radiographically, terminating in a thin wafer-like lamina on the sound tooth (Figs. 3, 5, 8, 9). In the author's opinion, this type of case is well suited to the use of bone implants. If, as seems possible, an adequate supply of mineral salts is what is required, bone of any of the three types used in this study should be suitable.

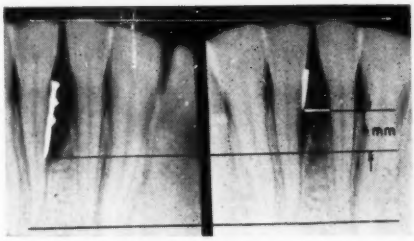

Fig. 8. Homogenous implant. Pocket before treatment, 8 mm., and sixteen and one-half months after, 4 mm.

Technique

A preliminary curettage is carried out at least two weeks before the proposed implantation, in order to remove all traces of deposits on the root surface. The author considers that it is wise, at this stage, to carry out soft-tissue curettage too, in order to eliminate granulations. The occlusion is checked, and the tooth concerned freed from any traumatic relationship. If there is more than slight mobility, the tooth is immobilized, or preparation is made for immobilization. It is considered advisable for the tissues involved to present minimal inflammatory change at the time of operation.

Fig. 7. Homogenous implant. Pocket before treatment, 12 mm., and twenty-seven and one-half months after, 4 mm.

The operation is carried out under local analgesia and penicillin cover—300,000 units of procaine penicillin intramuscularly. The shallower pockets—6-8 mm.—may, if limited to one aspect of the tooth, be treated by curettage with insertion of the implant into the curetted area, but in the great majority of cases it is necessary to raise a muco-periosteal flap. Before the flap is raised, the pocket epithelium lining is thoroughly curetted away, this being easier to perform before the flap is raised. After raising the flap, the pocket is curetted in order to remove granulation tissue, and the root surface is then planed with suitable hoes and curettes. The author prefers, for their small dimension and permanently keen edge, the new tungsten carbide-tipped hoe scalers recently described by him.[16] The intention is to remove all cementum. In a few cases, preliminary elective devitalization of the pulp and root filling is undertaken, as it is considered that curettage in the apical region would hazard the blood supply; in these cases, the cemental curettage is carried out at and around the apex (Fig. 15).

Small fragments or granules of bone are now inserted. In the case of ethylenediamine treated bone, this is made moist with a weak penicillin solution 400 units per c.c. The fragments are built up to, or

Fig. 9. Homogenous implant. Pockets before treatment, 6 mm., and 8 mm., and 6 months after, 2 mm. and 4 mm.

Fig. 10. Homogenous implant. Pocket before treatment, 10 mm., and almost twenty one months after, 8 mm.

Fig. 12. Homogenous implant. Pocket before treatment, 8 mm., and thirteen months after, 2 mm.

nearly up to, the highest level of the existing bone, but care is taken *not* to overpack. The adjacent tissues are freshened, if need be, to ensure an adequate blood clot.

The flap is then carefully sutured, using fine-gauge plastic sutures, and a piece of tin foil adapted to the area with a portion passing interdentally. Over this a surgical cement pack containing a small amount of Terramycin is placed. The tin foil is sterile and inert, and prevents the pack from forcing its way down the pocket into the operation area.

Post-operative penicillin is administered daily for four days; the pack and sutures are removed after a week. The patient is then carefully instructed in local oral hygiene, use of a soft brush for one week, careful use of interdental stimulators, avoidance of biting on the tooth concerned. No instrumental exploration is made for six weeks.

Results

Twenty-five of the 27 cases in this study have been re-examined by the author during February or March, 1957. Two cases were unable to attend, being abroad, and were assessed as at the last dates of examination, nine months and three months ago respectively. Pocket depth was noted and presence or absence of inflammation. Radiographs were taken with calibrated Hirschfeld silver points inserted; with few exceptions, this procedure was adopted before operation and these radiographs could be compared in respect of pocket depth and the relationship of the base of the pocket to the root. It is pointed out that the terminal portion of these points measures 4 mm., and the distance between the ridges 2 mm. In a few cases, silver points of 2 mm. are shown (Figs. 5, 6).

In a number of cases lines have been drawn parallel to the apices and incisal edges of the teeth involved and further lines parallel to these from the bases of the pockets before and after treatment. By measuring the silver point on the radiographs, it is possible to estimate *approximately* the degree of coronal shift of the

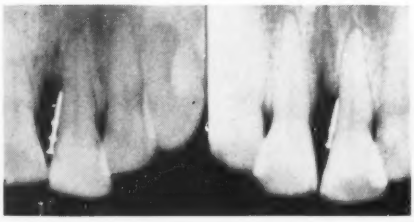

Fig. 11. Homogenous implant. Pocket before treatment, 8 mm., and fifteen months after, 6 mm.

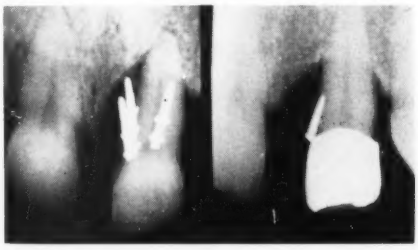

Fig. 13. Homogenous implant. Pocket before treatment, 8 mm., and thirty months after, 4 mm.

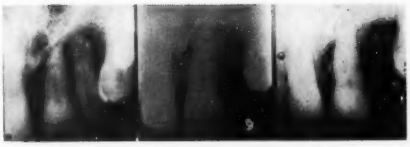

Fig. 14. Homogenous implant. Pocket before treatment, 10 mm., and thirty-one and one-half months after, 2 mm.

base of the pocket—it is not claimed that these measurements will be exact.

Cases which have exhibited, over a reasonable period of time, filling in of the bone defect or a substantial part of it, without necessarily showing the reconstruction of a lamina dura, a pocket of 4 mm. or less and freedom of the gingiva from inflammation or suppuration, were considered to be 'very satisfactory.'

Cases where only a limited amount of filling-in of the bone defect has occurred, and where a pocket of 6 mm. or less, was present, again with freedom from suppuration or gingival inflammation, were considered to be 'reasonably satisfactory.'

Cases with no bone regeneration, or where an implant had become completely resorbed and where pocket formation beyond 6 mm. persisted, were considered 'unsatisfactory.'

On this basis, the cases under review were classified as follows:

Very satisfactory	11.
Reasonably satisfactory	11.
Unsatisfactory	5.

The radiographs shown do not, of course relate to all the cases treated. The radiographs are immediately prior to the insertion of the implant, on the one hand, and of latest re-examination, on the other. In 5 cases, additional radiographs show the implant at some interim stage (Figs. 5, 12, 14, 15, 16). A case considered to be 'very satisfactory' is illustrated by Figure 5, 'reasonably satisfactory' by Figure 14, and 'unsatisfactory' by Figure 2. One case which was considered a failure was investigated histologically.† The initial condition (Fig. 2) was one of marked alveolar destruction between $\underline{/1}$ and $\underline{/3}$ but intrabony pocket formation was limited. The aim therefore, was to try to build up the crestal bone in this area. A splint was made, as the $\underline{/1}$ exhibited Grade 2 mobility, but it transpired later that the patient could not manage to eat with it, and had worn it very little. Three and a half months after the operation, at which homogenous (bone bank) bone had been used, suppuration was noticed from the pocket distally and the tooth was found to be non-vital. It was extracted, together with the interdental papillae and a small segment of bone.

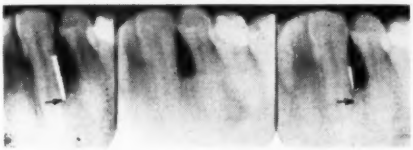

Fig. 16. Heterogenous (anorganic bovine) implant. Pocket before treatment, 8 mm. and about seven months after, 4 mm. N.B. alteration in position of pocket base as from kink in root (arrowed).

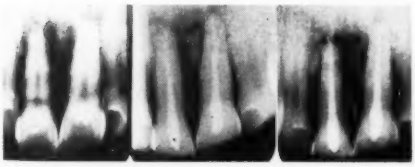

Fig. 15. Heterogenous (anorganic bovine) implant. Pockets 1/1 before treatment, 15 mm. and 14 mm. and four months after, 4 mm. and 4 mm.

There was found to be no connection between the tooth and this tissue, and it was therefore impossible to section the two together. The sections were stained with Haematoxylin and Eosin, and also with Mallory's connective tissue stain in order to demonstrate any new bone that might have formed (Fig. 17, 18). The pathologist's report was as follows:

†This case was reported to the British Society of Periodontology, at its meeting in February, 1957.

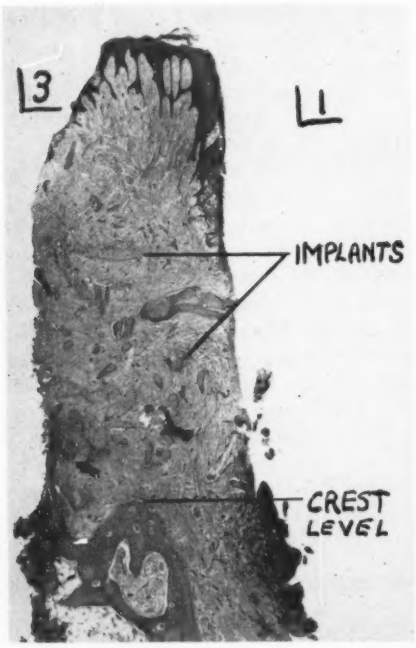

Fig. 17. Section through interdental bone and papilla adjacent to /1, (Fig. 2). Haematoxylin and Eosin, X13.

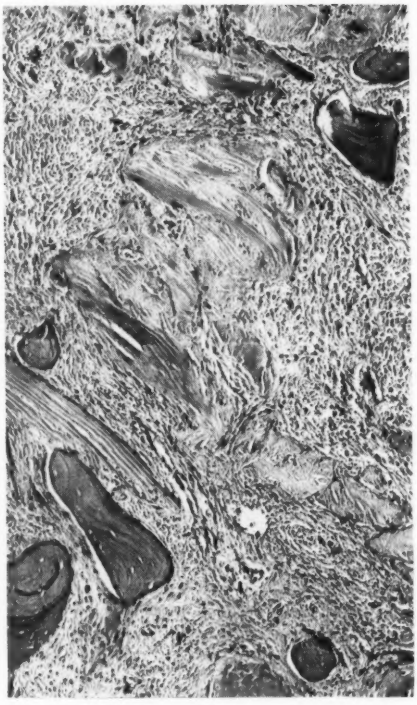

Fig. 18. Higher magnification of section shown in Figure 17. In a dense field of fibrous tissue, there are fragments of implanted lamellar bone with empty lacunae. There are also some areas of new bone formation, some being around the implants, some being discrete. These consist of coarse woven bone. No inflammation is seen.

Macroscopical—A roughly rectangular mass of interdental bone, height 12 mm. labio-palatal width 7 mm., mesiodistal thickness up to 5 mm. The superficial aspect is covered by interdental papilla. The mesial aspect is grooved to accept the root of the incisor.

Microscopical—The implanted fragments of lamellar bone are lying in dense and mainly inactive fibrous tissue. In some places a little irregular woven bone has been formed, sometimes on the surface of a fragment of implanted bone, sometimes between the implanted fragments. One of the most striking features of the specimen is the absence of osteoclastic activity; no osteoclasts are now present and there is little evidence of past resorption. The field of the implant is free from inflammatory cells, but there is a moderate degree of lymphocytic and plasma cell infiltration near the pocket epithelium.

This was fairly encouraging in that no inflammatory reaction was present around the implanted chips, and some new bone had formed in spite of the presence of suppuration. This is the only human histological material which the author has had the opportunity to examine at present.

SUMMARY

A review of the literature concerning bone implantation for periodontal diseases is followed by an account of the author's experiences over a period of five years. The technique of implantation is described, and the result of 27 cases, some of which are illustrated by radiographs, discussed. Of these cases, 3 were treated with autogenous, 20 with homogenous, and 4 with an-

organic bovine bone implants. The histology of the implant in one unsuccessful case is shown.

CONCLUSIONS

In appropriate selected cases, particularly those with intrabony pockets, bone implantation may be of considerable value. The author considers that care must be given to secure an operative field, free as far as possible, from chronic inflammation, to immobilize the tooth involved, if any mobility is present, to curette cementum thoroughly, probably removing it all, to remove all epithelium and to prevent infection.

ACKNOWLEDGMENTS

The author wishes to acknowledge his indebtedness to those of his colleagues in the Department of Periodontology, who have treated certain cases, to Captain Frank Losee, for kindly making available some ethylene-diamine treated bovine bone, to the Departments of Pathology and Photography at the Institute of Dental Surgery for the preparation of microphotographs and photographs, and the 'Dental Practitioner' for permission to reproduce certain radiographs of 4 cases which have been published previously in that Journal.

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Announcement

COURSE OFFERED BY BALINT ORBAN, M.D., D.D.S.

Dr. Orban's Course in Periodontics "Theory and Practice of Periodontics," will be given July 15th to 19th, 1957. Further information may be obtained by writing Dr. Balint Orban, 629 N. Nevada Avenue, Colorado Springs, Colorado.

Clinical and Roentgenographic Studies of Trauma from Occlusion*

by Ulf Posselt and Olle Maunsbach, *Malmö, Sweden*

THE theory of "excessive load on teeth" suggested by Károlyi[14, 15, 16, 17, 18] and Stillman's[31, 32] and McCall's[20] theory of "traumatic occlusion" are based upon clinical observations.

Histologic observations have been made to correlate functional stress and periodontal disease. Those of Box[4, 5] and of Häupl and Lang[13] indicated functional stress on the teeth to be the chief cause of advanced periodontal breakdown ("pericementitis fibrosa"; "paradentitis profunda"). Gottlieb[8, 9] and Gottlieb and Orban[10] regarded periodontitis as the cause of traumatic occlusion. Stones[35] claimed occlusal trauma to be a predisposing factor to periodontal disease.

Recent animal experiments[2, 7, 37] suggest that the changes occurring in traumatic occlusion are reversible and that only under extremely unfavorable conditions do they lead to a deepened gingival sulcus.[37]

The phrase "trauma from occlusion" in the present article is used to indicate the effect of injurious forces upon the teeth and no effort is made to differentiate between trauma from chewing or from bruxism.

SIGNS OF TRAUMA FROM OCCLUSION

A number of clinical[4, 25, 33] and roentgenographic[21, 22, 23, 24, 29, 30, 32] signs of "trauma from occlusion" have been enumerated. A great number of authors[3, 11, 12, 4, 16, 23, 25, 28, 33, 34, 36] have indicated mobility of teeth, observable at closure and during articulation movements, as a diagnostic criterion. This sign, which can be called "mobility from occlusion" is used extensively as a test for trauma from tooth to tooth contact.

Roentgenographic signs mentioned are widening of the periodontal membrane space,[21, 22, 23, 24, 29, 30, 32] thickening of the lamina dura,[29] hypertrophy of the cementum[21, 22, 23, 29, 32] and root resorption.

PROBLEMS INVESTIGATED

The purpose of the present investigation was to determine:

A. the percentage of individuals in the material showing one or more movable teeth;

B. the percentage of movable teeth among all teeth examined;

C. which group of teeth most often shows mobility;

D. the frequency of observable roentgenographic signs and the correlation between these and the mobility from occlusion.

The investigation was limited to one clinical sign, the mobility from occlusion. The four roentgenographic signs investigated were those previously listed as having been used in earlier writings.

It should be stressed that it was not the intention of the authors to investigate the significance of trauma as an etiologic factor in periodontal disease, nor was it an intention to develop or refine clinical or roentgenographic methods. During the study, the usual clinical and roentgenographic techniques were used.

MATERIAL AND METHOD

Ninety-seven male students, aged 20-29 years, with complete, or almost complete, natural dentitions were examined. Full mouth roentgenograms showed no periodontal bone destruction.

The individuals studied were selected from four different groups of dental students. In this way four different study

*From the Department of Occlusal Analysis and Rehabilitation. The Royal Dental School, Malmö, Sweden. Ulf Posselt is Head of the Department, and Olle Maunsbach Senior Instructor.

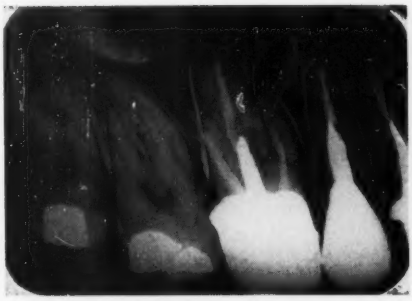

Fig. 1. Widening of the periodontal space. $\underline{5}$. $\underline{6}$. (Retouched)*

Fig. 2. Thickening of the lamina dura. $\underline{5}$, 4. (Retouched)

groups were obtained. The first two groups of 29 and 21 students were examined by Posselt and the other two consisting of 28 and 19 students by Maunsbach. Each subject was examined on two different occasions, after an interval of one week to 10 months.

Each examination consisted of (1) clinical inspection of the gingiva, (2) clinical tests for mobility from occlusion and (3) roentgenographic examination for the signs of trauma from occlusion.

The subjects were examined by placing the tip of the forefinger against the buccal surface of the tooth and palpating any movement during contact movements. Mobility from occlusion was examined (a) during biting into the occlusal (intercuspal) position, (b) small and (c) extensive lateral gliding movements.

For practical reasons, this examination was limited to the upper jaw. It included 14 upper teeth on the average per person. The result of the test was recorded as plus or minus, with no attempt being made to grade the extent of any mobility.

On analysis of the full mouth roentgenograms signs were recorded only when they could be clearly observed by both examiners.

*Editor's note: Author submitted unretouched photographs which demonstrated the same changes as shown here, but would not reproduce as well.

OBSERVATIONS

Referring to problem A—98 per cent of the subjects showed one or more teeth with mobility from occlusion.

Referring to problem B—altogether 1,349 teeth were examined 6 times each. Of 14 teeth on the average examined per person, 6 to 7 showed mobility. Posselt found 321 (46 per cent) of 698 teeth with clinical mobility. The corresponding figures for Maunsbach were 328 (about 50 per cent) of 651.

Referring to problem C—In both groups investigated the first bicuspid most frequently showed mobility from occlusion with an incidence of 72-78 per cent followed by the cuspids with an incidence of 66-72 per cent. The second bicuspids varied from 50-70 per cent. The incisors and the molars definitely showed less mobility than the bicuspids and cuspids. The cuspids seldom showed mobility from biting into the occlusal position.

It should be emphasized that this study was confined to the upper teeth. The mobility frequency sequence indicated should not be assumed to be identical in the lower jaw. On the contrary it is logical to assume that mobility patterns of the lower teeth would be the reverse of those in the upper teeth.

The results of the findings in two examinations in a group of 29 persons was studied

by the X^2 analysis. The ratio between the number of teeth that showed mobility on one occasion and the number that showed mobility from occlusion on both occasions was roughly equal when examined for the various mandibular movements.

Referring to problem D—Roentgenographic signs of trauma from occlusion, 80 teeth showed roentgenographic signs of trauma from occlusion (Figs. 1-4). Of these, 7 showed more than one such sign.

Among 649 teeth with mobility from occlusion, only 56 showed roentgenographic signs. Among 80 teeth with roentgenographic signs, 56 showed mobility from occlusion.

The second bicuspids most often showed roentgenographic signs. With regard to the correlation between roentgenographic signs and mobility from occlusion, the investigation showed that widening of the periodontal membrane space appeared simultaneously with mobility in 75 per cent of the teeth studied. Thickening of the lamina dura occurred simultaneously with mobility from occlusion in 73 per cent of the teeth studied.

DISCUSSION

It is true that the examination methods used in this paper are subjective, but no objective methods are available[1, 6, 19, 26, 29, 38] for measuring tooth mobility during contact movements.

From the literature one gains the impression that the signs of incipient periodontal disease are the same as the signs of trauma from occlusion.

According to a well known definition[34] "traumatic occlusion" is an "abnormal occlusal stress which is capable of producing or has produced an injury to the periodontium." Consequently, mobility from occlusion would imply that changes have occurred in the periodontal tissues which have caused or will cause periodontal disease.[31, 32]

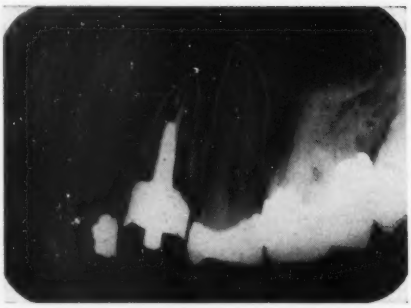

Fig. 3. Hypercementosis. $\underline{5}|$. (Retouched)

CONCLUSIONS

Mobility from occlusion was found in a great number of the persons examined. This fact indicates that one should be more hesitant in identifying trauma from occlusion by the clinical evidence of mobility from tooth to tooth contact.

Therefore, the following conclusions may be drawn: (1) for occlusal trauma to be diagnosed, the test of mobility from occlusion and roentgenographic signs is not sufficient and (2) the concept of trauma from occlusion must be further elucidated.

Whether the sign of mobility from occlusion has any diagnostic value is not made clear from the present investigation. Diagnostic methods in use should, however, be critically examined and discussed. Nothing can be built on illusions. Several of the

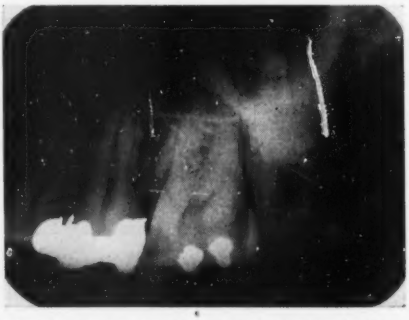

Fig. 4. Root resorption. $\underline{5}|$, $\underline{4}|$. (Retouched)

diagnostic techniques now in use no doubt rest on an insecure basis. It is, however, just as wrong to accept everything without criticism as it is to reject everything without first testing it. After critical examination, certain modifications might be made.

SUMMARY

Ninety-seven male students, aged 20-29, with healthy periodontal tissues were examined for mobility from occlusion. Palpation of the upper teeth was carried out on closure into the occlusal (intercuspal) position and during tooth articulation. Of the roentgenographic signs described in the literature as evidence of "trauma from occlusion," 4 were registered, namely widening of the periodontal membrane space, thickening of the lamina dura, hypercementosis and root resorption. (See Figs. 1-4).

Of 97 persons, with an average of 14 upper teeth, 95 had approximately 6 or 7 teeth with mobility from occlusion. This sign was observed most frequently among the first bicuspids, the cuspids, and the second bicuspids in the order listed.

Mobility from occlusion was seldom accompanied by roentgenographic signs, while roentgenographic signs were not always accompanied by mobility from occlusion.

It is stressed that caution should be exercised in the evaluation of trauma from occlusion, on the basis of the test for mobility from tooth to tooth contact. The concept trauma from occlusion requires further elucidation.

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Case Histories

by MYER D. WOLFSOHN, D.D.S., AND ROBERT S. WOLFSOHN, D.D.S., *Buffalo, N. Y.*

DENTISTS are often confronted with teeth that lie in strategic positions which appear condemned to extraction because of extensive periodontal involvement. These teeth may include some with over 50% bone loss, bifurcations, trifurcations cases, periodontitis complex and many other forms of pathology. Many of the teeth that fall into this category can be saved for long periods of time.

The retention of an anterior tooth is sometimes of great importance because of the replacement problem involved. This is also true of posterior teeth, especially where the mouth is affected with extensive periodontitis. It is frequently necessary to improvise special technics and surgery to attack some of these unusual situations but as a rule the many orthodox forms of treatment that are available today allow us to treat these cases successfully. The teeth described in this report are not all good long term risks, but with the exception of one experimental case, they are all functioning comfortably and the patients involved are in excellent health.

CASE 1. Mrs. S., age 37, had cystic like areas high up on the roots of the upper left cuspid, left central and right central incisor (Fig. 1). These areas were best approached through windows on the labial side, care being taken not to invade the apical areas. The top pictures (A and B) were made before surgery. The lower films (C and B) were taken ten months after surgery. Regular check up films have been made and the centrals are vital and all teeth in excellent condition at the present time.

CASE 2. Mr. H. S., age 72, had six upper anterior teeth supporting a partial denture (Fig. 2). He was most anxious to retain

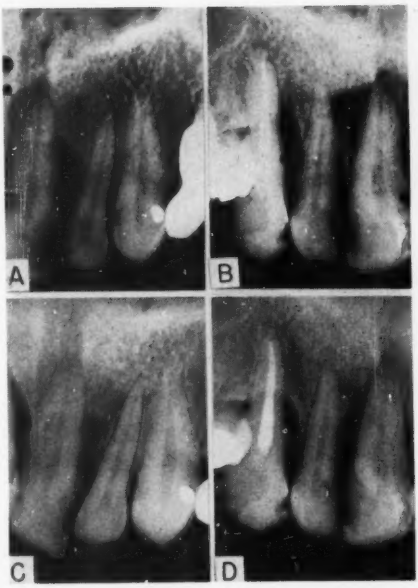


Fig. 1. Mrs. S., age 37 years. A. and B. show cystic like areas high on roots of left cuspid, left central and right central. C. and D. show same areas, after surgery, about one year later.

these teeth for obvious reasons. The upper right cuspid was vital but had extensive rarefaction and a deep distal pocket discharging free pus. Radiograph (A) taken 1-22-52, shows large rarefied area surrounding apical region. (B) taken 8-1-52, following subgingival curettage shows

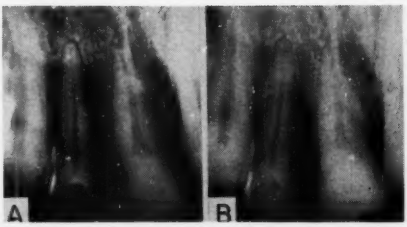


Fig. 3. A—Upper left lateral loose, sensitive and discharging pus. Flap operation performed. B—Almost five years later, tooth is firm, vital and free of pus.

some bone filling in with less discharge of pus. (C) taken 12-22-54. Some time after a flap operation shows area all healed and tooth very firm and vital. The 1 left lateral was found non-vital and root canal therapy was performed by referring dentist. The patient is now 76 years old and still retains these teeth.

CASE 3. Male 55 years old, with loose sensitive upper left lateral incisor discharging pus for many years (Fig. 3A). Flap operation performed shortly afterwards. (B), nearly five years later, shows tooth to be firm, healthy and vital and a very useful part of his upper dentition.

CASE 4. Miss L. B., age 30, had a periodontal abscess between lower incisor teeth (Fig. 4). This is not an uncommon condition and this case readily responded to surgery. Radiograph (B) shows consolida-

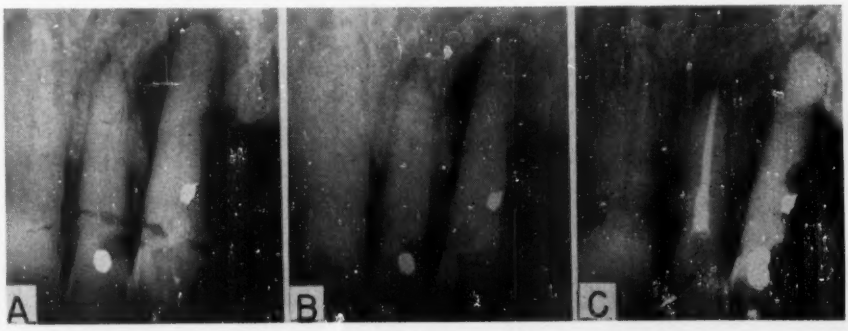


Fig. 2. Male, age 72 years. A—Upper right cuspid with extensive rarefaction and deep distal pocket discharging pus. Tooth vital. B—Some improvement following subgingival curettage. C—Several years later, after flap operation tooth still vital and firm, no pus.

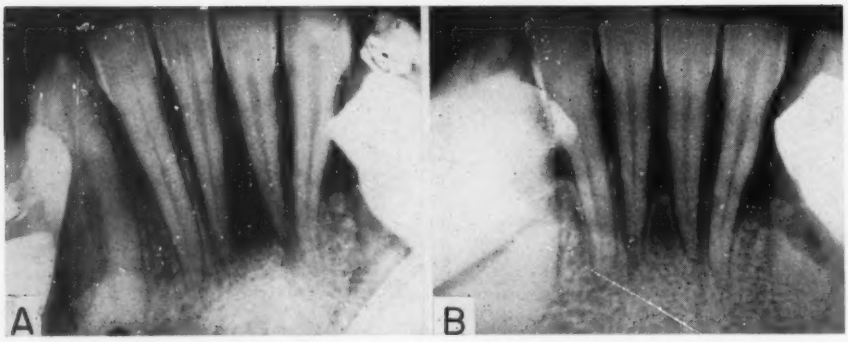


Fig. 4. Miss L. B. Age 30. A—Periodontal abscess between lower central incisors. B—One year later.

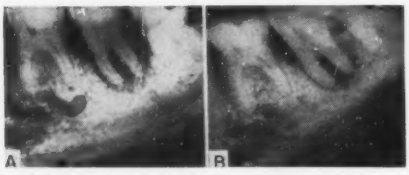


Fig. 5. Mrs. M. G. Age 47. A—10 mm. pocket on distal and 10 mm. on lingual. B—3 mm. pocket on distal and lingual pocket eliminated.

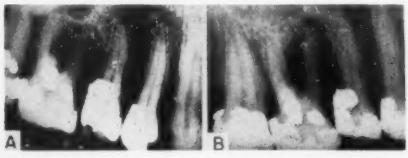
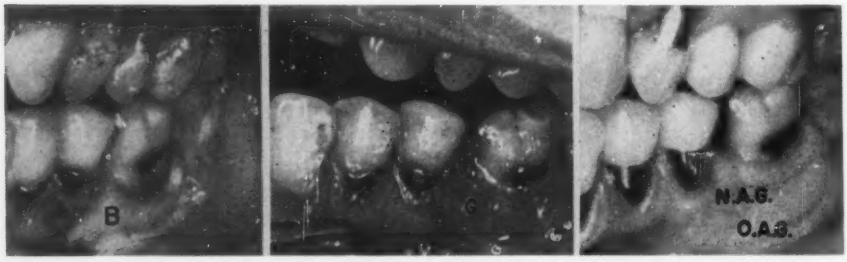


Fig. 6. A—Bone resorption and loss of matrix on mesio-buccal root of upper left first molar. B—A little over two months later shows a regeneration and filling in of bone matrix.

tion of bone matrix one year (after A).

CASE 5. Mrs. M. G., age 47, with a lower right second molar showing extreme case of periodontitis with 10 mm. pockets both on the lingual and distal surfaces (Fig. 5A). The tooth had normal vitality. This case was treated with antiformin over a period of time and showed excellent results (B two and a half years later). The tooth at that time revealed a 3 mm. pocket on distal and the pocket on the lingual eliminated. The patient had only two teeth affected by periodontal disease, the other one being the upper right second molar with a 10 mm. pocket on the buccal side. This tooth is not shown because the pathology does not show on the x-ray picture, but the pocket has been entirely eliminated using antiformin and curettage.

CASE 6. Female, age 55, insisted that she



Case 7. Illustration #1. Symbol B denotes exposed compact bone. **Illustration #2.** Postoperative picture 7/12/55. Notice granulation tissue "G." All bone is now covered by granulation tissue. **Illustration #3.** O.A.G. is the old and repositioned attached gingiva. N.A.C. is the new attached gingiva (eliminating the previous pocket) after 4 months.

wanted to have the upper left first molar extracted because it had previously given her much pain (Fig. 6A). Permission was given to treat this tooth and observe the results. The tooth showed much rarefaction around the mesial buccal root and in the trifurcation. Radiograph taken a little over two months later (B) showed that much of the bone matrix had filled in and the clinical picture showed no pockets. Several months later the tooth was sacrificed and examined clinically. It showed a distinct increase coronally in bone and periodontal membrane appeared to be normal in this section of the bone.

CONCLUSIONS

Although the foregoing cases might be considered as hopeless teeth, with good treatment, they can be maintained as effective abutment teeth temporarily; even redeposition of bone has occurred. This procedure tends to prolong the efficacy of the prosthesis, rather than remove all the teeth and use full dentures.—*175 Linwood Ave.*

Announcement

THE AMERICAN INSTITUTE OF DENTAL MEDICINE

The next Annual Meeting of the Institute will take place at The Oasis Hotel, Palm Springs, California, October 13-17, 1957. The faculty will consist of Dr. Edwin F. Alston, Clinical Instructor in Psychiatry at the University of California Medical School, who, because of the close relationship of many psychiatric and psychological problems to dental practice, will present a series of lectures in this field. Dr. S. J. Kreshover, Associate Director of the National Institute of Dental Research in charge of intramural dental research, will discuss selected subjects in the field of general and oral pathology. Dr. K. F. Meyer, Director Emeritus of the Hooper Foundation for Medical Research, the University of California, will discuss the exciting story of polio vaccine, and the advancement of scientific research in Russia. Dr. Max S. Sadove, Professor of Surgery, University of Illinois, has been highly recommended for his expert knowledge in the field of anesthesiology. He will correlate the various aspects of this subject to the practice of dentistry. Dr. Joseph F. Volker, Dean of the School of Dentistry of Alabama University, has agreed to speak about caries and fluoride and dental health. There will also be presentations by three of the younger research workers in the Division of Oral Biology, School of Dentistry, University of California, who are actively engaged in clinical and laboratory investigations: Dr. Sol Silverman, Dr. Theodore Grant, and Dr. Howard Myers.

All Seminar lecturers will participate in an Open Forum discussing the application of their subject to the practice of Dentistry. Because of the mounting interest in this annual meeting of the Institute, early registration is requested. The Institute also calls attention to the Case History Service which is furnishing Dental Medicine Case Histories with kodachrome slides, medical history, laboratory findings, roentgenograms, and all data pertaining to each individual case. Applications and full information concerning either the Annual Meeting or the Case History Service may be secured from the Executive Secretary, Miss Marion G. Lewis, 2240 Channing Way, Berkeley 4, California.

Guest Editorial . . . .

Role of Occlusal Forces

The problem of the role of occlusal forces in the field of periodontics is one of extreme interest and one in which we have relatively limited knowledge, yet have developed some hard shelled schools of thought. Every periodontal authority has his, or her, concept of acceptable articular relationships, and also of jaw to jaw or jaw to skull relationships, but by reviewing these various approaches to the problem it is quite obvious that there is a striking variance of opinion further confounded by confusion of terminology which makes me conclude that there is no one problem facing dentistry today greater than that of finding agreement on the questions of what is the correct jaw to skull relationship known loosely as centric relation, and what and where do practical acceptable articular relationships begin and end.

The question of the importance of overstress of occlusion as a causative factor in periodontal problems raises some interesting side issues that must first be considered and resolved.

First, what is overstress of occlusion? The old saying of, "what is one man's meat is another man's poison" certainly applies here. We know that two people can have the same physical experience, such as walking up a mountain in a snow storm and one will emerge exhilarated with a healthy glow, while the other will emerge exhausted and sick from the exposure. No two people react exactly alike.

Periodontosis, if it exists in a pure form, is said to be due to the inherent inability of the structures to withstand the forces brought to bear upon them. This is a vague and indefinite statement but probably will remain so until the amount of stress a tissue can stand under the various conditions of age, general health, environment, emotional imbalance, etc. are determined.

The condition of occlusal traumatism is a well accepted periodontal entity. In my mind this is a pure form of overstress in an otherwise healthy host. What would the effect be if this host already shows signs of other periodontal breakdown? It seems logical to suppose that the occlusal overstress is going to do much greater damage and become a causative factor in promoting further periodontal destruction.

The famous dog experiments done by Gottlieb and Orban and recently repeated by Orban and others showed no periodontitis or periodontosis, but a few dogs or monkeys cannot solve so complex a problem as this, for these animals had no deficiency diseases, no fatigue factors, no emotional factors, nor any other stress factors, all of which play an important role in the problems of the periodontal patient. This is a problem that will take painstaking controlled observation of large groups of human subjects over a long period of time.

I believe it is important therefore, to accept the point of view that overstress of occlusion is a factor in the development of periodontal disease, because if we say that it is not, all studies on the importance of overloading the periodontium will cease, and certainly further investigation is indicated.

Carlton H. Williams

Prize for Editorial

The William J. Gies Foundation for the Advancement of Dentistry, Inc. has established an annual award for the most valuable editorial written by a graduate dentist and published in a non-proprietary dental journal in the United States or Canada, on the subject of dental research, education, dental socio-economics, dental public welfare, interprofessional relationships, or dental organizations. The Foundation believes that in promoting such a competition it will serve the advancement of dentistry and carry out the wishes of Dr. Gies, who was particularly interested in these fields.

The initial judgment of editorials will be on those appearing in the 1957 volumes of the dental journals and the award to the writer will be in the form of a plaque citation. It will be accompanied by an honorarium in the amount of $200.

Editorials should be limited to 750 words and received by the Chairman of the Editorial Award Committee, Dr. John P. Traugott, 121 East 60th Street, New York City, by February 1, 1958. Participants are requested, whenever possible, to send only reprints or clippings, properly identified and bearing the name of the writer. It will not be feasible to return any of the material submitted. The winner of the award will be announced in the Spring of 1958.

AMERICAN BOARD OF PERIODONTOLOGY

The American Board of Periodontology announces that until January 1, 1962 it will examine acceptable candidates who have one academic year of formal graduate or post graduate education and training in periodontology and its supporting subjects and a second year of additional formal education and training in the field, or its equivalent. After that date only two academic years of formal education and training at an approved institution will qualify an applicant for the examinations of this Board. Three years of practice devoted principally and primarily to periodontics are in addition to the educational requirement in either case.

It is further announced that the date for filing applications is on or before July first of the year preceding that in which the examinations are to be taken.

Harold J. Leonard, D.D.S.
Secretary

DR. MARTHA JONES

We are sorry to report that Dr. Martha E. Jones, 515 David Whitney Building, Detroit, Michigan, died in December 1956. Dr. Jones was well known to most of the members of the Academy; her interest in periodontology dates back over thirty-five years. She was always a loyal supporter of all of the activities sponsored by the American Academy of Periodontology. Her practice was limited to periodontology.

The Infrabony Technique as a Predictable Procedure

by JOHN PRICHARD, D.D.S., *Fort Worth, Texas*

REGENERATION of bone that has absorbed due to periodontal disease is the ideal objective of therapy in advanced periodontal breakdown. Linghorne and O'Connell demonstrated regrowth of bone histologically in experimentally produced epithelized pockets in dogs.[1] Periodontists generally agree that regeneration is possible in the so-called infrabony pocket, but there is some doubt that the method will yield predictable results.

The purpose of this report is to show through documented case reports submitted in the sequence of attempted therapy that regeneration of bone does occur clinically following rational therapy for carefully selected infrabony pockets. The objective of this report is to demonstrate that the choice of favorable cases will yield successful results comparing favorably statistically with any other therapeutic modality. The topography of the bony deformity determines the therapy and the result that may be obtained.

Goldman and Williams established the infrabony pocket as a distinct entity in periodontal disease. An infrabony pocket differs from a gingival pocket only in that there is bone in its lateral wall, that is, the alveolar crest is coronal to the base of the pocket. Pocket elimination therapy also differs in that the pocket cannot be eliminated by reduction of edema or by removing the soft tissue wall through gingivectomy. Two methods are available for eliminating this type of pocket: (1) by inducing a new growth of bone and periodontium to fill in the infrabony defect, or (2) by removing enough crestal bone to eliminate the defect by creating a new level consistent with the general attachment level.[2]

While bone absorption shows mosaic individuality, definite patterns are common in each anatomic area because of the usual bone topography. Due to the width of the bone and the density of the cortical plates, the mandibular molar region is the most common site of the infrabony pocket. However, this type of bone deformity can occur on any tooth surface where bone is thick enough to have supporting bone between the alveolar bone proper and the cortical plate. Gottlieb[3] pointed out that this is not an intra-alveolar pocket, because the alveolar bone is destroyed as the pocket forms.

Etiology of the infrabony pocket is the same as that of any other periodontal pocket. Inflammatory processes causing pocket formation follow the peri-vascular channels of the nutrient blood vessels, destroying first the crestal bone, causing an initial cupping that is visible in the roentgenogram. After the inflammatory process has destroyed the crestal bone, the spongy bone will be absorbed more rapidly than the cortical bone which places the bottom of the pocket between the tooth and the

Fig. 1. Typical infrabony deformity of the mandibular molar region. There is a deep crypt like cavity with three bony walls surrounding the denuded mesial root.

Fig. 2 A & B. Maxillary premolar region, showing bony topography that is frequently found after absorption where the ridge is narrow. This deformity has one bony wall.

cortical plate, apical to the bone margin. If the alveolar bone proper is absorbed equally on approximating teeth, an interproximal bony crater will result instead of an infrabony pocket. This type of bone deformity has two bony walls, and it will rarely respond to therapy by a regeneration of bone. Osteoplasty must be done to reduce the buccal and/or the lingual cortical plate to eliminate the pocket.[4,5]

The infrabony poocket is frequently associated with the periodontal traumatic lesion which Goldman explains as follows: "If inflammation destroys the gingival fibers, and a pocket develops in an area where periodontal traumatism has destroyed the periodontal membrane in the crestal region, the epithelial attachment can migrate apically beyond the alveolar crest since there are no barriers."[6]

Definitive diagnosis demands a standardized roentgenographic technique. The most accurate picture of bone is obtained when a faint image of the soft tissue is visible on the film. The roentgenogram should always show the teeth in their anatomic position as found in the clinical examination. Where the teeth are approximately in normal alignment, the enamel caps and pulp chambers will be distinct and contacts will not overlap. Correct vertical projection superimposes buccal and lingual cusps of posterior teeth so that the occlusal surface is not recorded, only the tips of the cusps being visible on the film. The position of the marginal bone can be varied by the angle of projection, but an infrabony deformity can seldom be obliterated in even a grossly distorted roentgenogram. Even slight distortion is quite obvious upon careful observation. Careful probing is an essential part of the diagnostic procedure and surgical exploration is sometimes necessary to determine the exact topography of the bony deformity.

Figure 1 shows a mandible with an extensive infrabony deformity on the mesial and buccal of the first molar which follows the usual pattern of bone absorption in this region. Absorption has almost reached the apex of the mesial root and the bifurcation is exposed on the lingual aspect. On the buccal aspect absorption has extended well into the bifurcation. The buccal cortical plate is notched, but there is a deep cup-like defect inside the remaining cortical plate. Spongy bone is exposed inside this

Fig. 3 A. Exposed palatal bone showing an infrabony deformity, involving the trifurcation on the mesial surface of a maxillary first molar.

Fig. 3 B. After osteoplasty to eliminate the infrabony deformity.

area, giving the surface a sieve-like appearance. The defect has three bony walls with the tooth root as the fourth wall.

Following treatment of an infrabony deformity like the one illustrated by the dry mandible, regeneration of bone can occur to the level of the remaining cortical plate. Regeneration of bone is not expected in the bifurcation as shown here because this would be marginal elevation of bone which is not probable with present therapy.

Fig. 3 C. Appearance of the ginviva ten months after therapy. Sulcus depth is normal.

Figures 2 A and 2 B illustrate a pattern of bone absorption that occurs quite often where the bony ridge is narrow. This deformity has one bony wall which supports the second premolar. Three bony walls are a prerequisite for predictable regeneration of bone. However, therapy may result in an improved roentgenographic appearance due to greater density of bone following elimination of the inflammatory process. Regeneration of bone here would be a marginal elevation, not the filling in of an infrabony defect.

Infrabony pocket therapy aimed at reattachment follows the rationale presented by Goldman in 1949.[7] The suprabony gingival margin is excised; this tends to prevent the apical proliferation of the epithelium by increasing the distance it must cover and also eliminates the gingival portion of the pocket. Using the bone as a landmark, all granulation tissue, including the attachment and sulcular epithelium, is completely removed, being sure to reach the buccal, lingual, mesial, and distal extension of the absorbed area as well as the apical. The bone is used as a landmark; all soft tissue between the tooth and bone is removed; the bone is not curetted, but *treatment is directed at the bone deformity rather than the pocket.* The root surface is

Fig. 4 A, Case #1. A wide infrabony deformity on the distal surface of a mandibular cuspid. Therapy, 3/4/52.

Fig. 4 B. After therapy, 6/25/53.

carefully planed with sharp curettes and hoes. A flap approach is sometimes necessary for access, especially if the bone deformity is flask-shaped in the apical region.

Fig. 5 A, Case #2. Gutta percha point in a wide infrabony deformity involving the mesial and lingual surfaces of a mandibular first molar. Therapy, 8/14/52.

Fig. 5 B. After therapy 1/2/53. Sulcus depth of five mm. recorded with Hirschfield pocket marker. Gingivoplasty was done to eliminate the pocket.

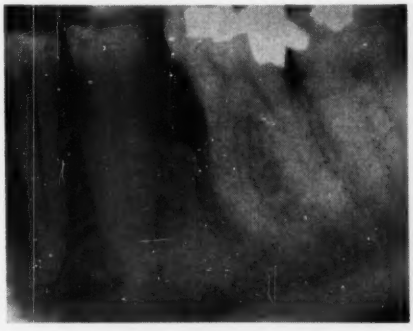


Fig. 5 C. 12/12/53. Sulcus depth is normal.

Fig. 6 A, Case #3. A wide infrabony deformity on the distal of the mandibular second molar. Therapy, 10/18/52.

Fig. 6 B. After therapy, 12/20/54.

Fig. 7 A, Case #4. A narrow infrabony deformity on the mesial surface of the mandibular second molar. Therapy, 10/15/53.

Fig. 7 B. After therapy, 3/18/54. Bone absorption instead of regeneration. Note the large trabecular spaces.

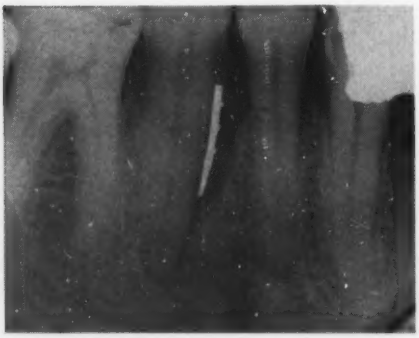


Fig. 8 A, Case #5. A deep marrow infrabony deformity on the mesial of the mandibular second premolar. Therapy, 10/29/53.

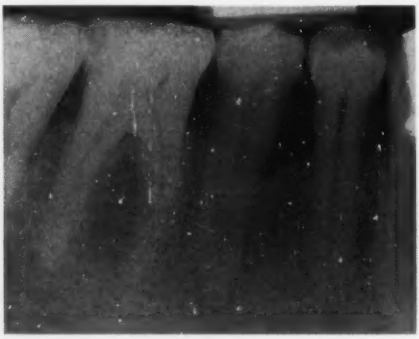


Fig. 8 B. After therapy, 3/12/55.

Fig. 9 A & B, Case #6. A wide interdental septum with infrabony deformities on the distal surface of the mandibular first molar and the mesial surface of the second molar. Therapy, 11/5/53.

Fig. 9 C. After therapy, 4/3/56.

Fig. 10 A, Case #7. Infrabony deformity similar to the one shown in the dry mandible, Fig. 1.

Fig. 10 B. Hirschfield pocket markers in the mesial, buccal, and lingual aspects of the pocket. Therapy, 11/7/53.

Fig. 10 C. After therapy, 6/16/55. Sulcus depth was six mm. on the mesial surface and a second stage procedure, including osteoplasty, was done.

Fig. 10 D. Flap retracted exposing bone for architectural reshaping. New bone has filled the infrabony deformity.

Before applying the periodontal surgical dressing, the embrasures and the buccal and lingual extensions of the pocket are covered with dry foil or Telfa[8] to prevent the dressing from squeezing into the pocket, thus displacing the clot.

The patient is routinely given a prescription for 72 hours dosage of an antibiotic because post operative infection would most certainly result in failure. The dressing is changed at weekly intervals for three weeks. Osteoplasty, when indicated to produce physiologic architecture, is usually done in conjunction with the infrabony technique, but a second stage procedure is sometimes necessary after bone regenera-

Fig. 11 A & B, Case #8. A 72 year old woman with an infrabony deformity in the distal surface of a mandibular first molar. Therapy, 11/10/53.

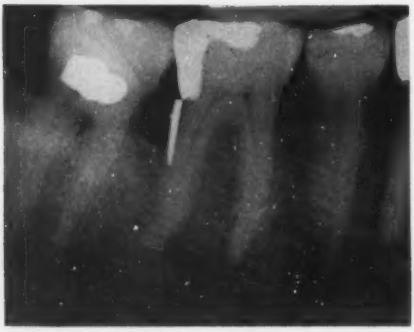


Fig. 11 C. 11/25/55 osteoplasty was done to eliminate a five mm. pocket that had recurred on the buccal surface of the distal root.

Fig. 11 D. 6/25/56.

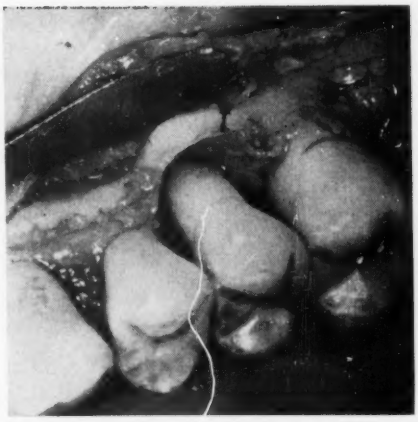


Fig. 12 A, Case #9. Flap retracted exposing infrabony deformity on buccal surface of maxillary second premolar. 10/4/54.

Fig. 12 B. The same area eight months later showing that bone has filled the infrabony deformity to within one mm. of the alveolar crest.

tion. If such etiologic factors are ignored, the process will reverse itself and the pocket and resulting bone defect will recur.

The occlusal factor is most important in these cases. The teeth involved must either be stabilized with provisional or permanent splints or be adjusted occlusally so that there is no trauma to disturb the healing process.

According to Ham, the blood clot does not appear to participate in the formation of new bone, but simply acts as a dressing protecting the wound. He has shown conclusively that in repair, new bone forms only from osteogenic cells that normally cover and line bone surfaces assisted by undifferentiated cells from the bone marrow.[9]

Success or failure can be determined by probing two months after treatment. Roentgenographically, the extent of bone formation can be seen in four months, but it is not well defined until six months after treatment. For critical evaluation of therapeutic results, the probe is indispensable.

The mesial surface of maxillary first molars often present a funnel-shaped deformity, infrabony in topography with the apex of the funnel pointing into the trifurcation, while the open end flares widely toward the palate. In the roentgenogram this may be seen in its beginning, as a shadow on the mesial surface, pointing to the trifurcation. Exploration reveals a wide crypt-like bone deformity. Re-attachment therapy is not usually successful in this situation and osteoectomy, as systematized by Schluger is the treatment of choice. Figure 3 A, B, and C.

The following case reports are submitted in the exact sequence of their attempted therapy with the results of treatment, both success and failure, described. The reason

Fig. 12 C. After osteoplasty.

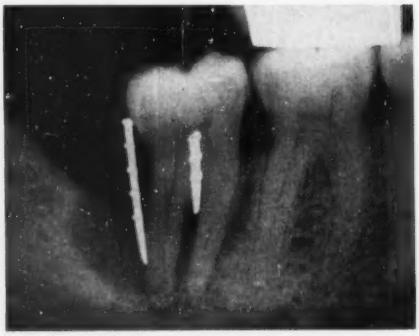


Fig. 13 A & B, Case #10. An extensive infrabony deformity on the distal of a mandibular second molar with bifurcation involvement. Therapy, 11/26/54.

Fig. 13 C. After therapy 2/17/56.

for this approach is to prove, if possible, that rational therapy on *carefully selected* cases will yield results that are as predictable as those of any other method used in periodontal therapy. The projection of diagnostic criteria is the objective.

Case #1, Figure 4 A, therapy 3/4/52—woman, age: 50. A wide infrabony deformity on the distal of the left mandibular cuspid. Fig. *4 B,* 6/25/53.

Case #2, Figure 5 A, therapy 8/14/52—woman, age: 28. A wide infrabony deformity on the mesial and lingual of the right mandibular first molar. Following curettment, the buccal bony wall was reduced in height due to a lack of confidence in regeneration. It was felt that the infrabony pocket

Fig. 14 A, Case #11. Infrabony deformity on mesial surface of mandibular molar, therapy 12/2/54.

Fig. 14 B. After therapy, 2/16/56.

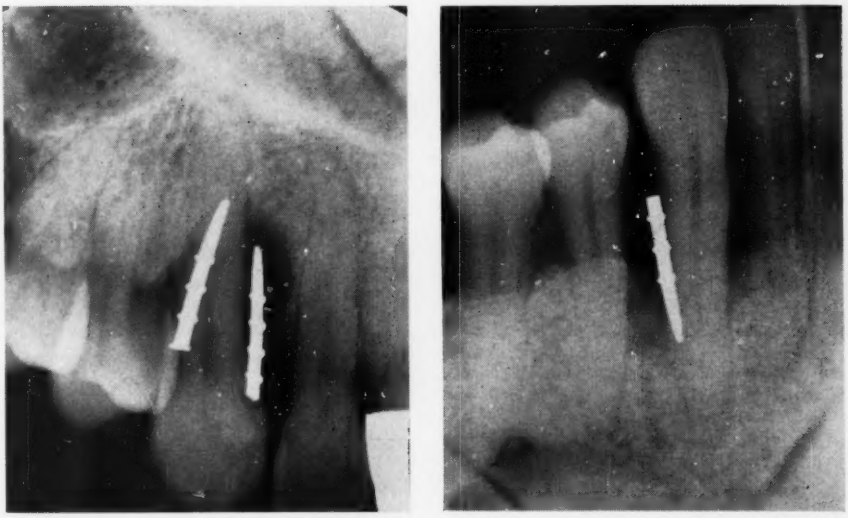


Fig. 15 A & B, Case #12. Maxillary and mandibular cuspids with infrabony deformities. Therapy 12/6/54.

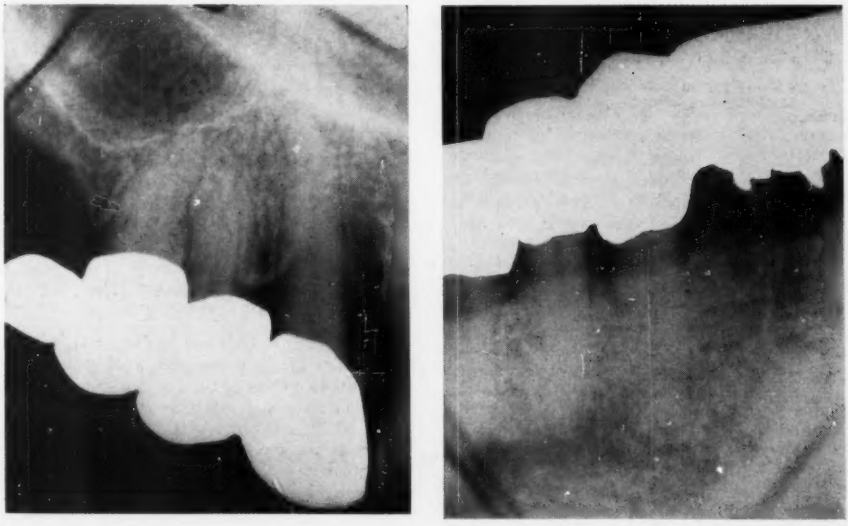


Fig. 15 C and D. After therapy 3/19/56. Periodontal prosthesis courtesy of Dr. O. W. Koberg.

Fig. 16 A, Case #13. Infrabony pocket on distal and lingual surfaces of mandibular second premolar.

Fig. 16 B. Therapy 1/10/55, following an acute excerbation of pocket in bifurcation of distal root of molar. Acrylic splint for temporary stabilization.

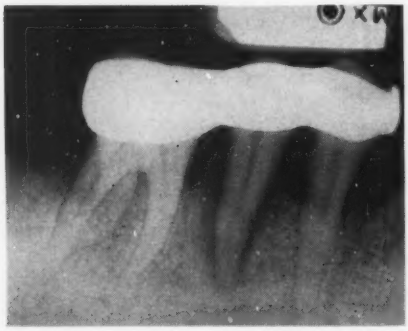


Fig. 16 C. Four months after therapy vitality tests were negative and endodontic therapy was done.

Fig. 16 D. 4/19/56.

was too wide to promise success. The tissue was sutured between the second premolar and the first molar. The result revealed a faulty diagnosis in the potential of regeneration. Five months after treatment, the sulcus was 5 mm. deep, *Figure 5 B.* This was reduced by gingivoplasty. *Figure 5 C,* 12/12/55.

Case #3, *Figure 6 A,* therapy 10/18/52—woman, age: 40. A wide infrabony deformity on the distal of the right mandibular second molar. *Figure 6 B,* 12/20/54.

Case #4, *Figure 7 A,* therapy 10/15/53—woman, age: 65. A typical infrabony deformity. Therapy resulted in complete failure with absorption of bone instead of regeneration. *The large trabecular spaces indicated a poor prognosis.* The age factor alone does not account for failure as treatment is often successful at advanced age levels. *Figure 7 B,* 3/18/54.

Case #5, *Figure 8 A.* therapy 10/29/53—man, age: 43. A deep narrow infrabony deformity on the mesial aspect of a left mandibular second premolar. *Figure 8 B,* 3/12/55.

Case #6, *Figure 9 A* and *B,* therapy 11/5/53—woman, age 46. There is a 10 mm. pocket on the distal of the right mandibular first molar; 7 mm. of this depth is infrabony. There is also a shallow infrabony deformity on the mesial of the second molar. Due to the distal position of the second molar a wide interdental septum is present between the molars. *Figure 9 C,* 4/3/56.

Case #7, *Figure 10 A* and *B,* therapy 11/7/53—woman, age: 50. Bone absorption in this case is almost identical to that of the dry mandible. *Figure 1.* The pre-treatment roentgenograms are shown in *Figure 10 B* with Hirschfield[10] calibrated pocket markers in the mesial, buccal, and lingual pockets. During curettage hemorrhage from the apex of the bone cavity had to be stopped with pressure packs of 1-1000 epinephrine saturated gauze. Further hemorrhage did not occur and a *clot was not formed;* during dressing changes, the wide sulcus was visible almost to its base near the apex of the mesial root. Bone regeneration occurred to the achievable optimum level in the usual period of time, *Figure 10 C.* However, the sulcus depth on the mesial was

6 mm., and a thick roll of gingiva caused soft debris to accumulate with resulting inflammation. A second stage procedure, *Figure 10 D*, including osteoplasty was done to eliminate the remaining pocket and produce a more physiologic gingival architecture. Restorative procedures are indicated to close the contact and improve coronal contours. This case and its behavior under therapy would seem to corroborate Ham's conception of bone repair.

Case #8, *Figure 11 A* and *B*, therapy 11/10/53—a 72-year old woman with an 11 mm. pocket on the distal surface of the left mandibular first molar. *Figure 11 C*, 10/25/55, a 5 mm. pocket with a purulent exudate developed on the distal buccal root extending into the bifurcation. The gingival wall of the pocket was excised and the buccal bone was ramped to a more ideal architectural contour. *Figure 11 D*, 6/25/56.

Case #9, *Figure 12 A*, therapy 10/4/54—woman, age: 40. The photograph shows a flap retracted with an infrabony pocket exposed on the buccal of a left maxillary second premolar. A thick buccal ledge of bone, extending from the cuspid to the second molar, had been reduced by osteoplasty with the exception of the area which was considered favorable for reattachment. The supporting and alveolar bone between the root and buccal cortical plate had absorbed, resulting in an infrabony deformity. Post-treatment probing indicated a successful closure of the pocket by reattachment and eight months later a second stage procedure was done to reduce the remaining bulge of bone for a more physiologic gingival contour. *Figure 12 B*, with the flap retracted, shows that bone has filled the space between the root and the cortical plate to within 1 mm. of the alveolar crest. This bone was reduced to a thin edge, *Figure 12 C*, and covered with a layer of Telfa which was then covered with periodontal surgical dressing, leaving the flap in its retracted position. This method of management, developed by Schluger and Fox, results in a wide band of attached gingiva and a knife edge free gingival margin.[11] This procedure can be done in a single stage operation, using the combined therapies of osteoplasty and infrabony technique where required.

Case #10, *Figure 13 A* and *B*, therapy 11/26/54—woman, age: 42. There is a large infrabony deformity on the distal of the second molar and a bifurcation involvement. The neighboring third molar, which was impacted, had been removed four years before. Infrabony pocket therapy was done and the enucleated tissue was submitted to histologic examination. The microscopic diagnosis indicated periodontitis, since chronic inflammatory elements pervaded the field. Two months after treatment a probe could not be inserted between the distal surface of the tooth and the gingiva. *Figure 13 C*, 2/17/56.

Case #11, *Figure 14 A*, therapy 12/2/54—a 65-year old woman with a 12 mm. pocket on the mesial of the right mandibular first molar. *Figure 14 B*, 2/16/56.

Case #12, *Figure 15 A* and *B*, therapy 12/6/54—woman, age: 48. Left maxillary and mandibular cuspids with infrabony deformities were stabilized with fixed periodontal splints. The upper cuspid was included as an abutment with the two premolars and a pontic about the size of a premolar was cantilevered from the distal of the second premolar, making a removable prosthetic appliance unnecessary. The mandibular cuspid was stabilized by inclusion in a fixed splint extending from the left second premolar to the right first molar, with pontics replacing the incisors and right second premolar. On the left side a third premolar pontic was cantilevered as above. *Figure 15 C* and *D*, 3/19/56.

Case #13, *Figure 16 A*, therapy 1/10/55—man, age: 48. This case presented a challenge. The right mandibular molars have been extracted and periodontal disease has caused bone absorption in the cuspid region. On the left side, the cuspid is missing; the second premolar has bone absorption almost to its apex on three surfaces. It could be elevated and depressed in its socket. The second molar is missing and the first molar has a bifurcated distal root with periodontal involvement. The first premolar would not be adequate as an abutment for a partial denture unless it was to be splinted to the other teeth around the arch to the right second premolar. Since this was not economically feasible for this individual, it was decided to attempt treatment before accepting a complete lower denture. A partial denture with clasps on the left molar and right second premolar was discarded.

At this time an acute periodontal abscess occurred on the distal of the first molar. While this caused a hyperemic destruction of bone in the intraradicular area,[12] *Figure 16 B*, it did not make prognosis for the molar less favorable.[13]

During therapy it was very difficult to hold the second premolar in position while planing the root surfaces. On one occasion, the tooth slipped out from under the retaining finger and moved a greater distance than the apical blood vessels could be expected to stretch. However, vitality tests were normal following treatment. An acrylic provisional splint was used for immediate stabilization which was replaced later by a cast vitallium band similar to the one described by Friedman.[14]

Four months after treatment the tooth was firm in its socket and bone regeneration had reached the horizontal level that was hoped for, but a wide and uneven periodontal membrane space remained. Vitality tests at this time were negative and endodontic therapy was done. *Figure 16 D*, 4/19/56.

Case #14, *Figure 17 A* and *B*, therapy 1/31/55—woman, age: 50. The interdental bone has absorbed equally between the right maxillary central and lateral incisors. There is a 12 mm. pocket on the distal of the central, but sulcus depth is normal on the mesial of the lateral. The curtain rod effect of the attachment on the lateral made it possible to treat the pocket as an infrabony deformity, since the more coronal attachment level maintained the

Fig. 17 A & B, Case #14. A 12 mm. pocket on the distal surface of the maxillary central incisor with normal sulcus depth on the mesial surface of the lateral incisor. Therapy 1/31/55.

architectural requirements for the infrabony technique. The result was more rewarding than expected, *Figure 17 C*, 4/30/56.

Case #15, Figure 18 A, therapy, 2/3/55—woman, age: 50. Extensive infrabony deformity on left mandibular cuspid. Treatment included stabilization with a fixed periodontal splint. *Figure 18 B*, 3/1/56.

Fig. 17 C. After therapy 4/30/56.

Case #16, Figure 19 A, therapy, 11/12/55—woman, age: 18. Infrabony deformity on mesial of left mandibular first molar. In the mandibular incisor region, horizontal bone absorption had reached the apices, rendering these teeth hopelessly involved; otherwise, bone and attachment levels were normal. *Figure 19 B*, 4/23/56.

Case #17, Figure 20 A, therapy, 3/12/56—man, age: 44, presented with an acute gingival involvement on the mesial and buccal of the left mandibular second molar. This tooth had tipped forward into the edentulous space, created by early loss of the first molar. In this type of deformity therapy for regeneration is not conducive to success because of the wide flaring form of the bony defect. Osteoplasty is indicated here.[15] *Figure 20 B*, 3/12/56, after osteoplasty and *20 C* 7/23/56, after restoration with a periodontal splint to prevent mesial drifting and give a consistent occlusal plane.

SUMMARY

The success or failure of the infrabony method depends to a greater degree on choice of cases to be treated than any other technique. This infers that diagnosis plays a greater part than in other methods.

The diagnostic criterion for a favorable result, i.e., the regeneration of bone to fill

Fig. 18 A, Case #15. Infrabony deformity on mandibular cuspid. Therapy 2/3/55.

Fig. 18 B. After therapy 3/1/56. Fixed periodontal splint courtesy of Dr. E. H. Hodges.

Fig. 19 A, Case #16. Infrabony deformity on mesial of mandibular first molar. Woman, age: 18, therapy 11/12/55.

Fig. 19 B. 4/23/56. Five months after therapy.

Fig. 20, Case #17. Wide flaring infrabony deformity on mesial surface of mandibular second molar. **A.** Before osteoplasty, 3/12/56.

Fig. 20 B. After osteoplasty, 3/12/56.

Fig. 20 C. 7/23/56 Fixed periodontal splint, courtesy of Dr. O. W. Koberg.

in an infrabony defect is the presence of three bony walls as a necessary topographic feature. This requirement is met most often in the mandibular molar region due to the width of the bony ridge and denseness of the cortical plates.

Stabilization of the involved tooth is also a necessary adjunct. This may be done on a temporary or permanent basis as the case requires.

CONCLUSIONS

Seventeen cases were presented in their chronological appearance for treatment with their success or failure in therapy shown. Four of these have been subjected to secondary surgical procedures, giving visual proof that new bone had filled in the infrabony defect. Certain diagnostic criteria have been put forward which appear to be definitive in success of treatment.—*4121 Camp Bowie Boulevard.*

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Dr. Cox reports that when a dentist's patient asks about fees, he should answer: "C your dentist twice a year."

A New Method for the *In Vitro* Production of Artificial Calculus*

by S. Wah Leung,† *Pittsburgh, Pennsylvania*

A RAPID, reliable and reproducible method for producing calculus under *in vitro* conditions simulating those in the human mouth is often desirable for the study of the mechanism of calculus formation and, more especially, for the evaluation of the effectiveness of possible calculus inhibiting agents which may be potentially harmful or unpleasant for use *in vivo*. A number of methods have been proposed and used in the past but some are suitable only for studies on a small scale,[1,2] while others require conditions which are so unnatural as to raise doubts concerning their applicability to human calculus formation.[3,4] In addition, these studies generally offer little or no conclusive chemical and crystallographic evidence that the deposits so formed are identical with natural calculus. The present paper reports a method by which calculus-like deposits may be produced rapidly and reproducibly on extracted teeth, and evidence is presented to indicate that these deposits possess many of the chemical and crystallographic characteristics commonly identified with natural calculus. The method is based upon the cyclic immersion and emersion of extracted teeth in saliva at body temperature and high relative humidity.

DESCRIPTION OF APPARATUS AND ITS USE

The apparatus consists essentially of a movable tray (Fig. 1) on which are placed the saliva containers, with the teeth suspended above the containers. The level of the saliva in relation to the crowns of the teeth is altered by merely tilting the tray to a set angle.

The tray is constructed of plastic or other suitable material, and made in the form of a low rectangular box with an open top. The four sides of the box extend about 1½ inches above the rim of the saliva containers when the latter are in position. The posterior border of the tray is attached by hinges to a horizontal bar mounted on two uprights of a height which would permit the tray to assume an angle of about 45° when the anterior border of the tray is resting on the bench top.

In order to alter the tilt of the tray from 45° to the horizontal, a wire cable is hooked to the front border of the tray, looped through a pulley suspended overhead, and finally attached to the rotating arm of a motor mounted below the bench on which the tray rests. The motor is geared to give one rpm. When in operation, the motor, through the cable and pulley, lifts and lowers the front part of the tray, thereby effecting a change in the tilt of the tray from the resting 45° position to the horizontal, and back, during each rotation. By suitably adjusting the amount of slack in the wire cable when the tray is in the resting position the proportion of each cycle during which the tray will be at rest, and the proportion when it is being either lifted or lowered may be easily controlled. We have arbitrarily set this ratio to ½ cycle when the tray is resting at the 45° angle, ¼ cycle for the raising and ¼ cycle for the lowering. In other words, during each cycle of 1 minute the tray is at the 45° for ½ minute, being raised during ¼ minute and being lowered during the remaining ¼ minute.

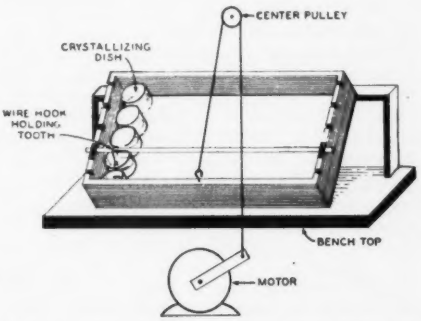

Fig. 1. Diagram of **In Vitro** Calculus Producing Apparatus. Only 5 saliva containers are shown in position. The tray is hinged to the horizontal strip at the back.

The saliva containers used are Pyrex cylindrical

†University of Pittsburgh School of Dentistry Department of Physiology.

*Based on a paper presented at the 31st General Meeting of the International Association for Dental Research, March 20-22, 1955, Philadelphia, Pa. Financial support for this study was generously provided by Johnson & Johnson, New Brunswick, N. J.

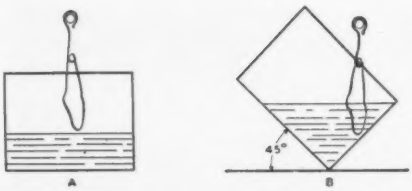

Fig. 2. Detailed drawings showing the change in the relative positions of the tooth crown to the saliva when (A) the tray is lifted to the horizontal position and (B) when the tray is resting at the 45° angle.

crystallizing dishes, 2" diam. x 1½" high. These are placed in 5 rows of 10 each on the tray. Routinely, 20 ml. of saliva are used in each container. With the saliva dishes in place and the tray in the horizontal (raised) position the height of the teeth is adjusted so that the crowns of the teeth are out of the saliva, with the tips of the cusps barely touching the surface of the liquid (Fig. 2A). Now, when the front part of the tray is lowered to the resting (45°) position the flow of the saliva toward the lower or front portion of the containers raises the level at this point and the crowns become immersed in the fluid (Fig. 2B). Thus, with each cycle, the crowns will be immersed for about ½ minute, exposed to air for about ¼ minute, and in the process of being either immersed or exposed for the remaining ¼ minute. Roughly, then, the crowns are either completely or partially submerged in saliva during 2/3 of the time and exposed to the atmosphere during the remaining 1/3 throughout the experimental period. Comparative tests showed this alternate immersion and emersion to be superior to a method which depends on the continuous submergence of the teeth in saliva for the development of calcareous deposits on tooth surfaces.

To hold the teeth in position a polyethylene-covered wire hook is inserted through a small hole drilled in a mesio-distal direction through the root of each tooth close to the apex. The other end of the hook is looped over a glass rod which is firmly fixed at both ends to the sides of the tray by means of spring clips. These glass rods are positioned so that they lie parallel to the rows of dishes, one to each row, and slightly towards the anterior half of the containers (Fig. 1). The teeth are suspended so that the crowns are directly over the saliva containers, and the distance between each tooth and the container is adjusted so that, when the tray is tilted, the crown will be completely submerged in the saliva (Fig. 2B). We have found two teeth for each container to be a convenient number. With fifty containers (5 rows of 10 each) a total of 100 teeth may be used at the same time. By testing a different agent with each group of 5 to 10 containers several agents may be simultaneously studied under identical experimental conditions. As a matter of convenience we have generally tested each agent on 20 teeth at a time (10 containers), with 10 or 20 teeth as controls. This enabled 4 agents to be tested at the same time under the same conditions. In the control teeth, grossly visible amounts of deposit will be formed at the end of two weeks and amounts sufficient for routine chemical analyses by the end of 4 weeks. During each experiment the entire apparatus is placed in a constant temperature room regulated at 37°C., with the relative humidity adjusted to 60-70%. The high humidity is necessary to prevent excessive evaporative loss of fluid from the open containers.

PREPARATION OF THE TEETH

The teeth to be used should be non-caripus, without fillings, and must be thoroughly cleaned and the pulps removed beforehand. Single-rooted teeth are preferred. The root tip is sliced off, the pulp completely removed with a broach, and the canal reamed to make certain no residual pulp tissue remains. The purpose of this is to prevent putrifaction of the pulp during the experiment. Following removal of the pulp the surface of the crown is thoroughly cleaned with a slurry of pumice, a small hole drilled through the root near the apex in a mesiodistal direction, and the tooth stored in distilled water until used.

COLLECTION AND USE OF THE SALIVA

Thus far we have used only stimulated saliva as the substrate for these experiments. Paraffin stimulated saliva was collected from students each morning of the school day, an average of 25-30 ml. from each of approximately 50 students. In the beginning we kept the saliva from each student separate throughout each experiment, always using the same student's saliva for the same teeth. In later experiments the saliva was pooled before use, providing a more uniform substrate with which to work.

Following collection and pooling of the saliva the pH was determined and the saliva distributed in 20 ml. aliquots to clean saliva containers. In instances where chemical agents are to be tested these were added to the required amount of the pooled saliva before the latter was placed in the individual containers. The containers were then set up on the

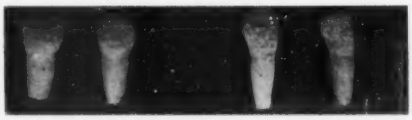

Fig. 3. Gross appearance of teeth with synthetic calculus incrustations. Coating the teeth on the right with silicone SC77 prior to the experiment was ineffective in reducing or inhibiting deposition.

tray, the teeth mounted in position, and the cyclic immersion started and continued until the next change of saliva. The saliva was changed every 24 hours during each day of classes, and 72 hours over the week-end. Each day, after removal of the old saliva and before setting up the fresh saliva the teeth were carefully sprayed with a stream of distilled water from a wash bottle for the purpose of removing any loosely adhering material, such as mucinous plaques and films.

At the end of the experiment the teeth were removed, cleaned with a stream of distilled water, allowed to dry in air, and then, if required, scored for the relative amounts of deposit present. The scoring was made on the basis of a 5 point scale, from 0, indicating no deposition, to 4, representing very heavy incrustation. For a more accurate determination of the quantity of deposition the synthetic calculus was scaled from each tooth, pooled with deposits removed from other teeth of the same group, dried and weighed. Chemical and other analyses may then be made on the removed material.

RESULTS

The gross appearance of the teeth after 4 weeks' treatment with plain saliva is shown in Figure 3. The 2 teeth on the right had been given a coat of silicone preparation before being subjected to the saliva treatment; the 2 on the left had not. As may be seen the deposit is fairly evenly distributed over the tooth surfaces. It is somewhat more granular and less well condensed than clinical calculus as found in the mouth. This may possibly be attributed to the absence of compressive action by the tongue and muscles of the cheek during the formation and deposition of the *in vitro* material. In spite of this the deposit is firmly adherent to the surface and may be removed only with difficulty. Of course, it should be remembered that any loose material would have been removed by the daily spraying with distilled water mentioned above. The granular nature of this "calculus" may be seen from the photomicrograph taken under low power with oblique surface illumination (Fig. 4).

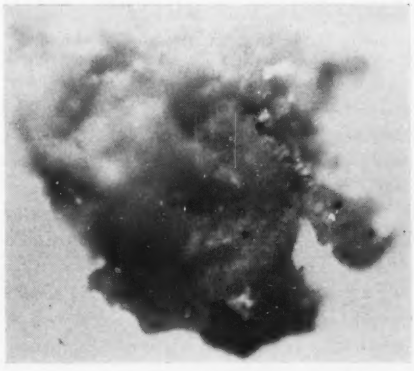

Fig. 4. Low-power photomicrograph showing the surface texture of the synthetic calculus.

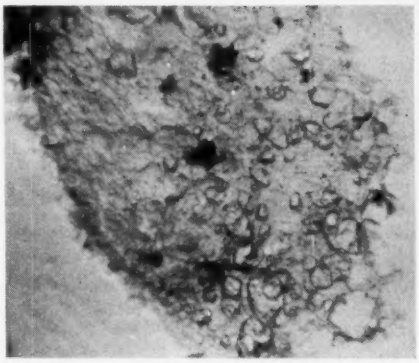

Fig. 5. Low-power photomicrograph of synthetic calculus showing the organic remnant after the inorganic material had been removed by acid decalcification.

As with clinical calculus the artificially formed deposit is composed of inorganic salts deposited on an organic matrix. This matrix is revealed after the inorganic components have been removed by acid, as shown in Figure 5. The nature of this organic material has not been determined.

In order to establish more precisely the similarity or dissimilarity of the *in vitro* material to clinical calculus, samples of the material from several experiments were analyzed chemically and compared with analyses of clinical calculus as reported in the literature (Table 1). The major difference between these two sources of deposits appears to be the higher organic and lower inorganic content of synthetic material. In other words, the *in vitro* "calculus" was not as highly mineralized as the clinical calculus. On the other hand, the relative proportions of these two major fractions

TABLE 1

Composition of Synthetic and Clinical Calculi

Constituent	Synthetic Calculi Sample				Clinical Calculi (from literature)
	A	B	C	D	
H_2O+Organic (%)	—	—	—	51.8	13.0—26.0
Organic (% dry basis)	31.2	32.0	31.8	—	12.9—28.4
Inorganic (% dry basis)	68.8	68.0	68.2	—	71.6—87.1
Inorganic (% wet basis)	—	—	—	48.2	—
Ca (% Inorg. basis)	31.1	—	—	43.1	30.7—39.0
P (% Inorg. basis)	15.2	—	—	20.1	16.8—20.5
Ca/P (weight basis)	2.04	—	—	2.14	1.79—2.16
CO_2 (% Inorg. basis)	—	—	—	3.8	0.28— 7.4

in the synthetic material remained remarkably constant from one experiment to another, done separately over a period of many months. Also, it will be noted that there is a marked similarity in the percentage composition of the major inorganic constituents between the natural and the synthetic material. Thus, the values for calcium, phosphorus, calcium/phosphorus ratio, and carbonate in the synthetic material are in good agreement with the reported values for clinical calculus.

According to a number of published reports the principal crystalline form of calcium and phosphorus in calculus is that of apatite, especially hydroxyapatite, similar to that found in enamel, dentine, and bone.[5,6,7,8,9] The presence of whitlockite and brushite has also been reported by several of these authors. In order to further establish the extent of similarity between the synthetic and clinical calculi, samples of material from two experiments were sent to Dr. Otto R. Trautz, New York University School of Dentistry, who very kindly subjected them to x-ray diffraction analyses for us. The pertinent parts of his report are quoted: "Sample 8-A, 1/20/54. Brown powder, gives a diffuse apatite diffraction pattern comparable to that obtained from dentine or bone. Sample 86-B, 1/20/54. Brown powder, gives a diffuse apatite pattern. The 211, 112, 300 reflections are slightly separated, indicating that the apatite is a little better crystallized than in sample 8-A. . . . Also, on account of the diffuse character of the apatite pattern one cannot say more than that the peak positions in 86-B are similar to those of OH-apatite." Thus, from both chemical and x-ray diffraction studies, it is clear that, at least in those characteristics considered to be important distinguishing features of calculus, synthetic material and the naturally occurring calculus are similar.

CONCLUSION

A method is described for developing calculus-like deposits on extracted teeth. The method is simple to use, is suitable for studies of the mechanism of calculus formation as well as for evaluating on a large scale potential calculus-inhibiting agents under carefully defined and controlled conditions. The results are reproducible from one experiment to another, and the deposits formed by this method have been shown to be closely similar to naturally occurring calculus on the basis of chemical and x-ray diffraction analyses.

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Announcement

XII CONGRESSO INTERNAZIONALE DI ODONTO-STOMATOLOGIA

From all parts of the world are coming enrollments to the XIIth International Dental Congress of the F.D.I., taking place at the Congress building of the E.U.R. in Roma, from 7th to 14th September, 1957.

The scientific programme comprises 25 reports and 150 co-reports covering all fields of dentistry: Anatomy and physiology, anesthesia and surgery, caries and protecting therapy, periodontal diseases, radiology, prosthesis, dento-maxillo orthopedics, social dentistry. Before September 7th, 1957 all reports will be published in English with a summary in French, German, Italian and Spanish in the International Dental Journal, and in Italian in the "Rivista Italiana di Stomatologia." In their September and October issues both Reviews have already published the reports of J. F. Walsh (Otago, New Zealand), J. G. de Boer (Groningten, Netherlands), J. A. Salzmann (New York), C. G. Paffenbarger (Washington), H. H. Stones and F. A. Lawton (Liverpool) and E. Müller (Hamburg, Germany).

By means of simultaneous translations Congress members will be enabled to hear the reports, co-reports, and discussions in the five official languages, i.e. English, French, German, Italian and Spanish.

Hundreds of demonstrations displayed by medical dentists from all over the world, a great number of films, and television transmissions will serve to make known the most modern methods, apparatus, materials, etc. particularly useful in daily practice. During the Congress days there will take place also the meetings of the International A.R.P.A. and those of the Armed Forces and many panel discussions.

Healing of Human Periodontal Tissues Following Surgical Detachment from Non-Vital Teeth

by MELVIN L. MORRIS, B.S., M.A., D.D.S.,* *New York, N. Y.*

MANY histological and clinical studies have recorded observations on the healing between periodontal tissues and roots following their separation by surgery or accidental trauma. Two experimental conditions are prominent among those that may influence this healing. The first is communication with the gingival crevice, introducing the possibility of infection or invagination of an epithelial barrier. The second is the status of tooth vitality since death would certainly affect the participation of a tissue in a healing reaction. These two factors serve as a convenient method of classifying past work.

CONNECTED WITH GINGIVAL CREVICE

A. *Vital*—Surgical wounds created in both man and animals[20, 29, 33, 41, 43, 45, 49] have generally shown connective tissue healing against curetted and uncuretted cementum and dentin with the formation of new cementum, the periodontal fibers generally running parallel to the root but assuming a functional orientation with longer healing periods.[33, 39] Similar results are noted when surgery is apical to a naturally occurring periodontal pocket.[34] Stones[43] reported contrary results in monkeys where the epithelial lining grew past denuded dentin, union starting only when cementum was reached.

B. *Non-Vital*—Waerhaug[45, 46] showed a section from a healed surgical wound about a human tooth with a gangrenous pulp. Healing occurred against both cementum and dentin, new cementum forming in a deep terminal dentinal nick.

C. *Reimplanted Teeth*—These have been given a separate grouping since tooth vitality becomes questionable at the instant of complete severance from the body. These wounds are, of course, in communication with the gingival crevice.

Evaluation of this work is difficult because of experimental variations affecting vitality, such as delay until reimplantation, sterilization methods during this period and the status of the root canal contents.

All variations of resorption, osseous union and atypical connective tissue unions have been reported.[19, 23, 24, 30, 38, 47, 48] Immediate reimplantation has produced the most favorable results.[12, 31]

REMOTE FROM GINGIVAL CREVICE

A. *Vital*—An orderly reconstruction[3, 4, 5, 6, 7, 8, 35, 40] of cementum, periodontal membrane and bone has been traced from less specialized connective tissue elements, following intentional trauma.

The majority of accidental root fractures studied[13, 21, 25, 26, 37] have shown new cementum covering widely separated or uniting closely apposed fragments. However, new dentin, bone or bone-like substances[15, 32] have also occupied the same role.

B. *Non-Vital*—Apicoectomies,[1, 10, 16, 18] up to fourteen years post-operatively, showed new cementum growing from the undisturbed root sides but covering only small portions of the denuded dentin. A connective tissue capsule covered the remainder of the exposed dentin. Experimental wounds on a non-vital tooth[40] showed similar results.

Root canal therapy without apicoectomy[17, 18, 22, 42] has shown cementum growing into and often occluding the apical foramen when the canal is slightly under-

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Presented before the Philadelphia Section of the International Association for Dental Research November 6, 1955.

filled. Cementum fails to cover material projecting beyond the apex.[22, 42]

It was obvious that information was scarce about surgical wounds connected with the gingival crevice on non-vital teeth. The present study attempts to fulfill this need.

CLINICAL PROCEDURE

The study was conducted on periodontally sound non-vital teeth, condemned as useless for prosthetic reasons. A tooth was considered non-vital if it had a root canal filling, an old non-bleeding pulp exposure with a periapical radiolucency, or a periapical radiolucency with negative results on the electric pulp tester. Labial surfaces, devoid of clinical inflammation, were chosen for ease of access and the relative frailness or total absence of overlying bone. These factors lent themselves to simplicity of procedure, minimal patient discomfort, and absence of deformity following extraction with adjoining tissues.[33]

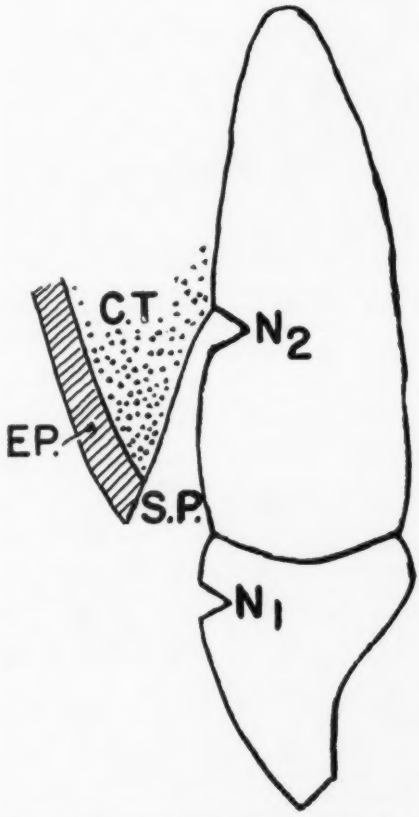

Fig. 1. N_1—Coronal Nick. N_2—Nick at the Most Apical Point of Surgical Separation. S.P.—Surgical Pocket. C.T.—Connective Tissue. EP—Epithelium.

Surgical wounds, 2-3 millimeters wide, were created by inserting a narrow scapel in an apical direction between gingivae and root. In some instances, thin labial bony plates were separated from the root with a Wiedelstadt chisel. Landmarks were made with a #2 wheel bur on the labial enamel and at the depth of the surgical separation (Fig. 1). Conditions were varied by curetting cementum, removing it or leaving it untouched. The clot was protected by gauze for ten minutes and healing then allowed to progress without further interference.

Measurements were made from the enamel nick to the gingival margin and point of soft tissue attachment before and after surgical separation.

Biopsies were performed in the following manner (Fig. 1). Two labial vertical incisions were made, one at each side of the healed surgical pocket. The two incisions were then joined about two millimeters apical to the estimated end of surgical separation. Modifying the techniques of Workman[49] and Box,[14] the bony trabeculae included in the operated area were separated from the remainder of the alveolar process by following the lines of incision with a #35 end cutting diamond disc, cutting into the root to insure complete isolation. To avoid trauma to the clinging tissue, the tooth was delivered labially with a straight elevator applied to the palatal surface of the root.

The extracted masses were fixed in Bouins' Solution, decalcified, imbedded in paraffin and stained with hematoxylin and eosin. Seventeen teeth were operated on.

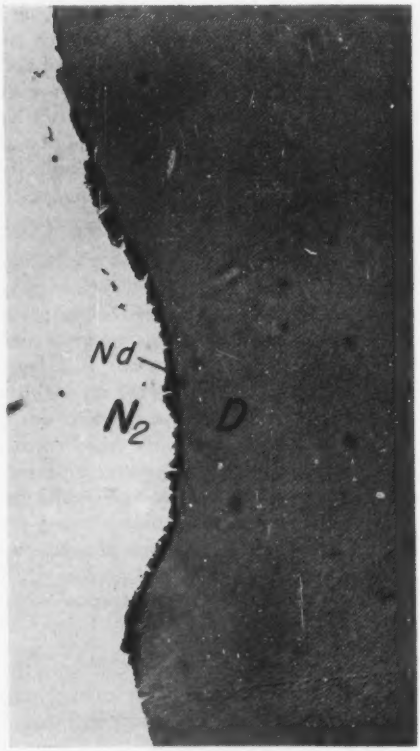

Fig. 2. Necrotic material (Nd) on dentin (D) of tooth with root canal filling.

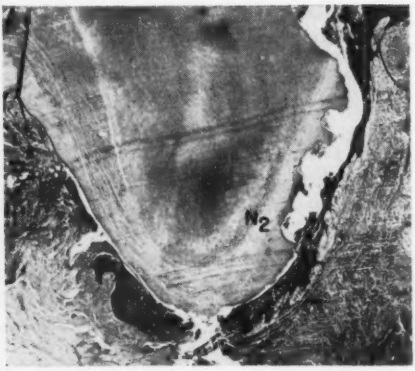

Fig. 3. Extension of pocket with infection (CI) present. Epithelial lining (EL) has passed beyond terminal nick (N_2).

Twelve biopsies were obtained after healing intervals ranging from 14 to 157 days.

RESULTS

A. Clinical—Utilizing the figures in Table 1, the results may be summarized as follows:

a. Apparent Healing
   1. Two showed no healing
   2. One showed healing of one-half millimeter
   3. Four showed healing of 1.0 millimeter
   4. One showed healing of 1.5 millimeters
   5. Three showed healing of 2.0 millimeters
   6. One showed healing of 4.0 millimeters
   7. Five showed a loss of periodontal connection

b. Level of Gingival Margin
   1. Eight showed no recession
   2. Two showed a recession of 0.5 millimeter
   3. Three showed a recession of 1.0 millimeter
   4. Two showed a recession of 1.5 millimeters
   5. Two showed a recession of 2.0 millimeters

B. Histological—Tooth #1 (Fig. 2) is a 71 day specimen with a root canal filling whose cementum was curetted. Inspection shows that cementum was actually removed during the curettage. The apparently attached soft tissue fell off the specimen during processing. Necrotic debris (Nd) is seen on the dentin (D) in nick N_2.

Tooth #2 (Fig. 3) is a 70 day specimen with a chronic carious exposure, and large, well defined apical radiolucency. Cementum was removed from the coronal half of the wound. N_2 marks the apical end of surgical separation.

The epithelial lining (EL) extends around the apex of the root into the bifurcation where the pocket communicates with an area of chronic inflammation marked by an intense round cell infiltration (CI). Other sections of the connective tissue also show intense cellular infiltration.

Tooth #4 (Fig. 4) is a 157 day specimen with an apical radiolucency. This tooth responded negatively to the electric pulp tester. The cementum was removed down to the nick (N_2). A is artefact where tissue was torn in sectioning. Coronal to N_2 resorption bays (R) may be seen on the dentin (D) filled with a light pink staining material. Adjacent to this is connective tissue (CT) with fibers running parallel to the root surface.

The epithelial lining (EL) and epithelial attachment (EA) are noted separated from dentin (D) by artefact (A). The connective tissue supporting the epithelial lining shows a heavy cellular infiltration (CI).

Figure 4A is a higher magnification of the area of dentinal resorption (R) showing union with connective tissue (CT) with fibers parallel to the root surfaces.

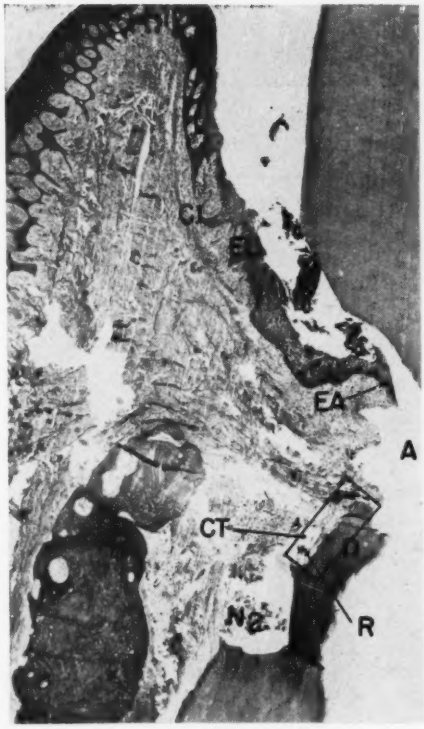

Fig. 4. Tooth with periapical radiolucency and negative pulp test showing attachment on dentin (D) coronal to N_2. Cellular infiltration (CI) is noted beneath epithelial lining (EL).

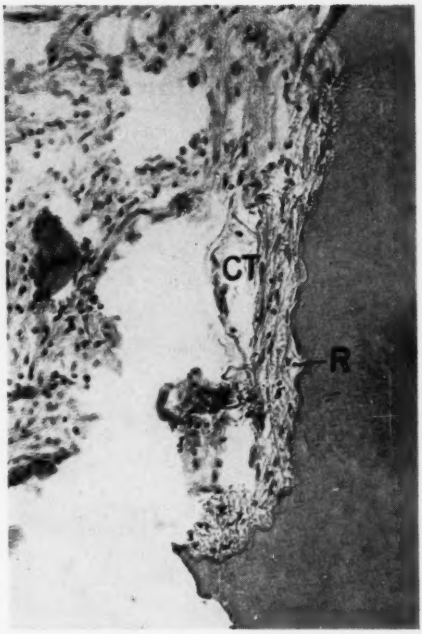

Fig. 4A. Inset of Figure 4 magnified. Resorption bays (R) are seen with adjacent connective tissue (CT) parallel to root.

Tooth #6 (Fig. 5) is a 42 day specimen with a root canal filling, whose cementum was curetted. Nick (N_2) may be observed penetrating deeply into dentin (D). Concavities (R) are noted on the cementum (C) which are either resorption bays or curette gouges. A dark border of connective tissue faces the exposed dentin (D) in N_2. Pieces of cementum (C) resulting from the experimental procedure are seen in the space adjacent to the alveolar bone (AB). Figure 5A is a higher magnification of the area about N_2. The connective tissue (CT) (which appeared darker on lower power) in direct contact with dentin (D) appears compressed with flattened cells parallel to the root surface. A light pink staining material enclosing cells, resembling new cementum (NC) covers the cut ends of the cementum (C) at both apical and coronal sides of N_2. A formation resembling immature bone (X) is seen in the connective tissue protruding into N_2. Figure 5B is a higher magnification of N_2 taken from a plane at one side of N_2 (see Fig. 5C). Although artefact separations (A) have occurred it is evident that new cementum (NC) is present at the apical end of N_2 from cementum (C) to the depth of the nick.

Tooth #8 (Fig. 6) is a 28 day specimen with a chronic carious exposure and with a periapical radiolucency. In addition, a fistula ran from the root apex to the surface of the labial gingivae. The cementum was removed from the coronal half of the wound exposing dentin (D) leaving some cementum at C_1. The terminal nick (N_2) may be observed only partially penetrating the cemental surface (C). Figure 6A is a higher magnification showing the termination of the epithelial attachment (EA) at the junction of exposed dentin (D) and cementum (C). Multinucleated cells (MC) are noted to the right of this area.

Tooth #10 (Fig. 7) is a 47 day specimen with a root canal filling with cementum undisturbed. The biopsy procedure inadvertently removed the terminal nick. Cementum shows numerous resorption bays (R). The coronal tip (AB_1) of the alveolar bone (AB) appears to have been fractured, probably by the experimental procedure. A dense cellular infiltration (CI) is noted in the stroma under the epithelial lining (EL) of the crevice.

Tooth #11 (Fig. 8) is a 35 day specimen with a chronic carious exposure and periapical radiolucency. The cementum was partially removed from the coronal half of the wound. The epithelial lining

CHART I

Case No.	Tooth No.	Fig. No.	Tooth	Status of Pulp	Pre-Operative Measurements (Mm.)			Mm. Surgical Separation
					N_1 to Soft Tissue Attachment	N_1 to Gingival Margin	N_1 to N_2	
1	1	2	/ 1	R.C.	1.5	—	6.5	5.0
2	2	3	4 /	A.	—	2.0	7.5	5.5
3	3	—	/ 2	A.	3.0	3.0	10.0	7.0
4	4	4	1 /	B.	2.0	0.5	5.5	3.5
5	5	—	2 /	R.C.	3.0	1.0	5.5	2.5
	6	5	3 /	R.C.	2.5	0.0	5.0	2.5
	7	—	1 /	R.C.	4.0	2.5	7.0	3.0
6	8	6	/ 2	A.	3.0	0.0	7.0	4.0
	9	—	/ 3	A.	5.0	0.0	7.0	2.0
7	10	—	2 /	R.C.	3.0	1.0	5.0	2.0
8	11	8	1 /	A.	6.0	1.0	7.0	1.0
9	12	—	/ 2	A.	7.0	5.0	10.0	3.0
10	13	9	3 /	A.	6.0	3.0	11.0	5.0
11	14	10	/ 1	R.C.	6.0	3.0	9.0	3.0
12	15	11	/ 3	R.C.	1.0	0.0	5.0	4.0
13	16	12	/ 3	R.C.	2.0	0.0	5.0	3.0
14	17	13	3 /	R.C.	2.5	0.0	6.0	3.5

R.C.—Root Canal Filling Present. A—Chronic Carious Exposure with Periapical Radiolucency. B—Periapical Radiolucency with Negative Pulp Test.

CHART I

Days of Healing	Post-Operative Measurements (Mm.)				Remarks
	N_1 to Soft Tissue Attachment	N_1 to Gingival Margin	Recession	Apparent Healing	
71	2.5	—	0.0	4.0	Cementum curetted
70	6.5	—	0.0	1.0	Coronal half of cementum removed
63	9.0	4.0	1.0	1.0	Coronal half of cementum removed, tissues torn in extraction
157	4.0	1.0	0.5	0.5	Coronal half of cementum removed
42	4.0	1.0	0.0	1.5	Cementum curetted, no biopsy
42	4.0	2.0	2.0	1.0	Cementum curetted
42	10.0	3.0	0.5	—3.0	Cementum curetted, no biopsy
28	0.0	7.0	0.0	0.0	Coronal half of cementum removed
28	0.0	9.0	0.0	—2.0	Coronal half of cementum removed, no biopsy
47	5.0	1.0	0.0	0.0	Simple surg. wound
35	9.0	1.0	0.0	—2.0	Coronal half of cementum removed
28	8.0	6.0	1.0	2.0	Coronal half of cementum removed, tissue torn in extraction
49	9.0	5.0	2.0	2.0	Coronal half of cementum removed
42	8.0	3.0	0.0	1.0	Coronal half of cementum removed
35	3.0	1.0	1.0	2.0	Coronal half of cementum removed
14	6.0	1.5	1.5	—1.0	Coronal half of cementum removed
21	8.5	1.5	1.5	—2.5	Cementum curetted

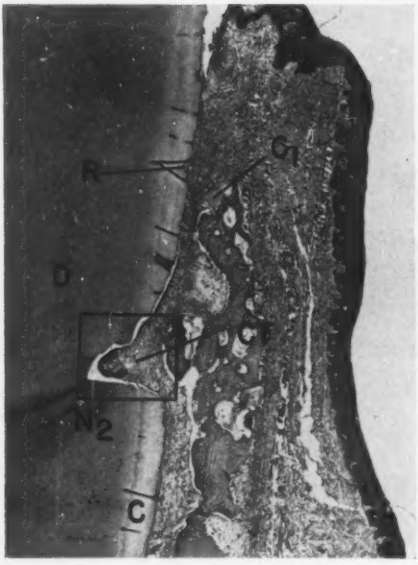

Fig. 5. Tooth with root canal filling whose cementum was curetted. The connective tissue (CT) in the nick (N_2) shows a dark border facing the dentin (D). A fragment of cementum (C_1) is noted near resorption bay or curette gauge (R).

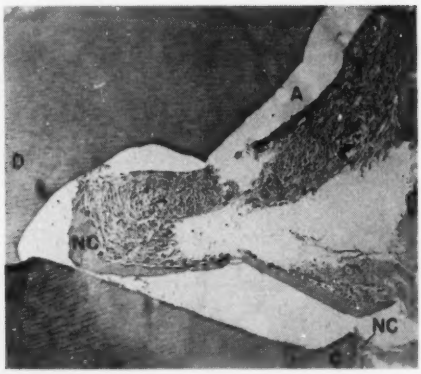

Fig. 5B. Another magnification of inset of Fig. 5 taken from a plane at one end of N_2 (see Fig. 5C). New cementum (NC) is noted lining most of the nick.

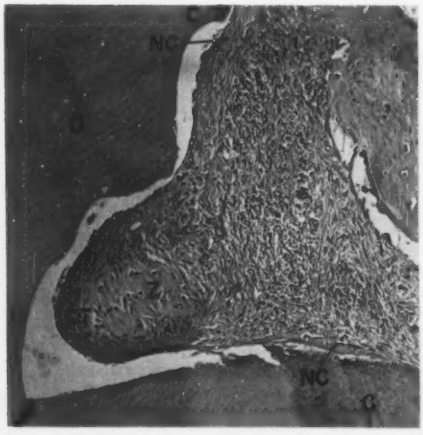

Fig. 5A. Inset of Fig. 5 magnified. The dark border of compressed connective tissue (CT) is noted facing dentin (D). A structure resembling immature bone (Z) is seen. New cementum (NC) is observed growing across the cut ends of old cementum (C).

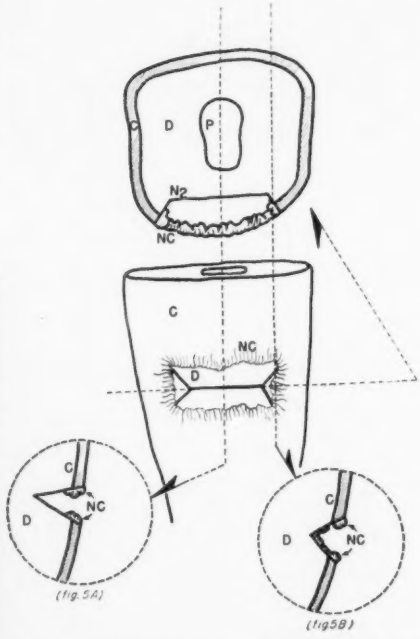

Fig. 5C. Diagrammatic representation of the planes of sectioning of Figures 5A and 5B. Figure 5A was taken from the mesiodistal center of the root and shows new cementum crawling in from the coronal and apical sides. Since the nick is a three dimensional entity, a section taken from the mesial or distal end (Fig. 5B) shows cementum covering the deeper recesses. This latter cementum has simply grown in from the lateral border of the defect.

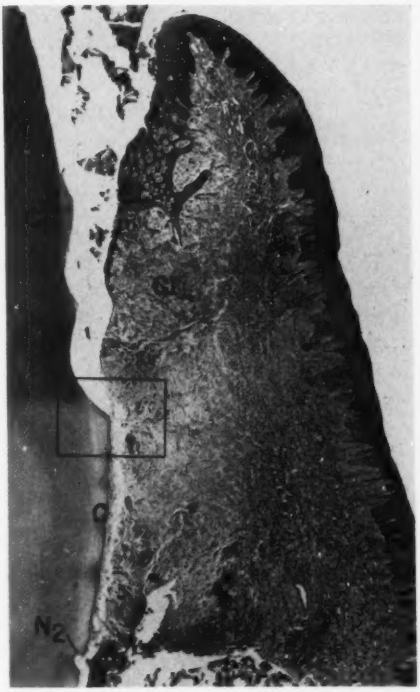

Fig. 6. Tooth with chronic pulp exposure, periapical radiolucency and fistula. Some crevice cementum (C_1) remains. Attachment begins with remaining cementum (C). A cellular infiltration is seen beneath the epithelial lining (EL).

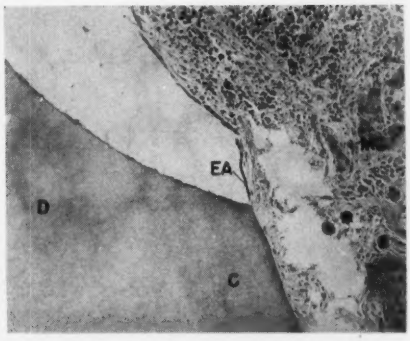

Fig. 6A. Inset of Fig. 6 magnified. The epithelial attachment is located where cementum (C) appears. Multinucleated cells (MC) appear in the neighboring tissue.

(EL) extends past untouched cementum (C) to the epithelial attachment (EA) which is located at the level at which the cementum has been mechanically removed. The epithelium near the point of attachment (EA) shows signs of hydropic degeneration and the underlying stroma shows a mild cellular infiltration (CI). Figure 8A, a higher magnification, shows the connective tissue (CT) in close contact with cementum (C) and dentin (D) in resorption bays (R) with fibers parallel to the root.

Tooth #13 (Fig. 9) is a 49 day specimen with a chronic carious exposure and periapical radiolucency. The coronal portion of the cementum was removed. Processing artefacts (A) have destroyed the epithelium. The terminal nick (N_2) was partly invaded in the biopsy procedure.

New cellular cementum (NC) may be seen in the terminal nick (N_2). A cellular, calcified material (NC_1) is present, which may be new cementum torn from the root, or alveolar bone. Figure 9A, a higher magnification of N_2 shows new cellular cementum (NC) deposited on dentin. A cellular pink cementoid material (X) is seen adjacent to old cementum (C) directly coronal to N_2. Figure 9B, a higher magnification of the area of denuded dentin (D), shows highly compressed connective tissue (CT) with flattened cells clinging to the dentin, the fibers being parallel to the root. Clear cementoid (X) is also noted on the other side of artefact (A).

Tooth #14 (Fig. 10) is a 42 day specimen with a root canal filling whose cementum was removed from the coronal half of the wound. The epithelial attachment (EA), separated from the root by artefact (A), is near the junction of exposed dentin (D) and cementum (C). Surgical artefact (SA) has accidentally obliterated the terminal nick. Resorption bays (R) are noted on the cemental surface. The adjacent connective tissue fibers (CT) are parallel to the root surface.

Tooth #15 (Fig. 11) is a 35 day specimen with a root canal filling whose cementum was removed from the coronal half of the wound. The terminal nick (N_2) has also exposed a portion of the dentin (D) apical to cementum (C). A is shrinkage artefact. Clear pink staining material (X) is seen covering the cut end of the cementum (C_1) at the apical end of N_2 and extending on to the dentin (D). A remnant of the torn epithelial lining (EL) is noted, with an adjacent inflammatory cellular infiltration (CI). AB is alveolar bone. Figure 11A is a higher magnification of the apical end of N_2 showing the deposition of new material (X) over old cementum (C_1) onto the dentin (D). AB is alveolar bone. Fragments of tooth substance (T) may be seen in the connective tissue protruding into N_2.

Tooth #16 (Fig. 12) is a 14 day specimen with a root canal filling whose cementum was ostensibly removed from the coronal half of the wound. Inspection shows, however, that cementum (C) remained up to point Y.

Hemorrhage (Hb) from the biopsy procedure is noted at the gingival margin and apical to N_2 at the biopsy artefact (SA). The epithelial attachment (EA) is located at approximately the junction of old

Fig. 7. Tooth with a root canal filling. The terminal nick was removed by the biopsy procedure. The coronal tip of bone (AB_1) appears to have been fractured in the experimental procedure. A cellular infiltration (CI) is noted under the epithelial lining (EL).

cementum (C) and the denuded dentin (D). A cellular infiltration (CI) is noted below the epithelial attachment. The connective tissue border (CT) facing the root across artefact (A) appears to be compressed with fibers and cells parallel to the root. Fragments of cementum (C_1) resulting from the experimental procedure appear in the connective tissue.

Tooth #17 (Fig. 13) is a 14 day specimen with a root canal filling whose cementum was curetted. The terminal nick (N_2) has not penetrated to dentin (D). A is processing artefact. Hb is hemorrhage resulting from the biopsy procedure.

The labial gingiva has been lost in sectioning. The epithelial attachment (EA) ends where some of the inner layers of cementum (C_1) remained.

The deeper sections of the connective tissue show a dense cellular infiltration (CI). The cementum at the apical end of N_2 is either a spur or has been distorted in processing.

DISCUSSION

Clinical—In the present study, 10 out of 17 teeth showed clinical healing. Five of these exhibited healing of 1 mm. or less. Since a periodontal probe with millimeter markings has an error of at least 0.5 mm.,[34] these become unreliable proofs of healing. Thus, 5 out of 17 teeth evidenced clinical signs of apparent healing.

The identical procedure performed on vital teeth[33] resulted in healing in 10 of 11 teeth. Only one healing area was as small as 1 mm., a case in which a mere 2 mm. wound had been originally produced.

These differing results may be partly explained by the fact that healing occurs against both dentin and cementum of vital teeth,[33] but not against the dentin exposed in the coronal half of the wound adjacent to non-vital teeth (Fig. 2, 6, 6A, 10, 11, 12, 13).

Previous papers displayed complete correlation of histological and clinical data with regard to healing[33] and to absence of healing.[34] Such correlation was lacking in the present paper and presented itself in two ways. Teeth #1 and #2, for example, were thought to have undergone healing until the histological pictures (Fig. 2, 3), were examined. Teeth #8, 11, 16 and 17 (Fig. 6, 8, 12, 13), on the other hand, showed more healing histologically than would be expected from the pre- and post-operative clinical measurements. Such discrepancies could be explained if the probe plunged through epithelium into the connective tissue. These four teeth were all examined in post-operative periods of less than five weeks. It is possible that such early healing tissue might be weak enough to permit the invasion of a blunt instrument, If this were true, it would also throw doubt on the validity of the other measurements in this paper. Although the above discrepancies were not observed about vital teeth,[33] easy tearing in sectioning of healing periodontal tissues was noted by Stones[43] and in a previous[33] and the present study. Whether healing periodontal tissues about non-vital teeth are truly weaker or different in some other way than those around vital teeth could only be determined by more extensive studies. It is interesting to note, in this regard, that proprioceptive changes in periodontal membranes following root canal

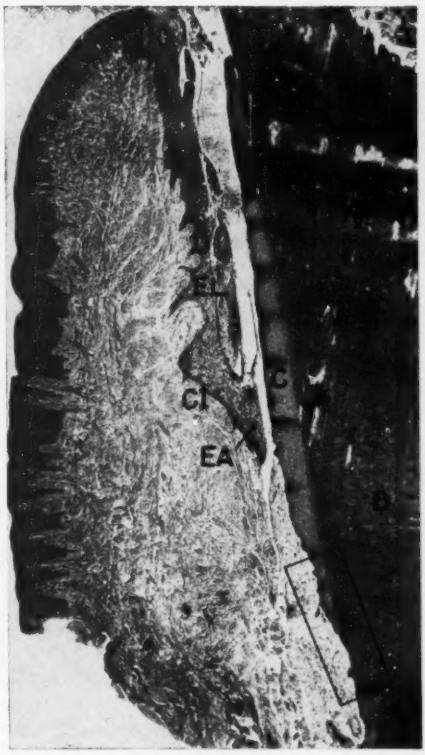

Fig. 8. Tooth with chronic carious exposure and periapical radiolucency. Cementum (C) was removed from the bottom of the pocket. The epithelial attachment (EA) is located at the point where cemental removal began. A cellular infiltration (CI) is noted in the connective tissue.

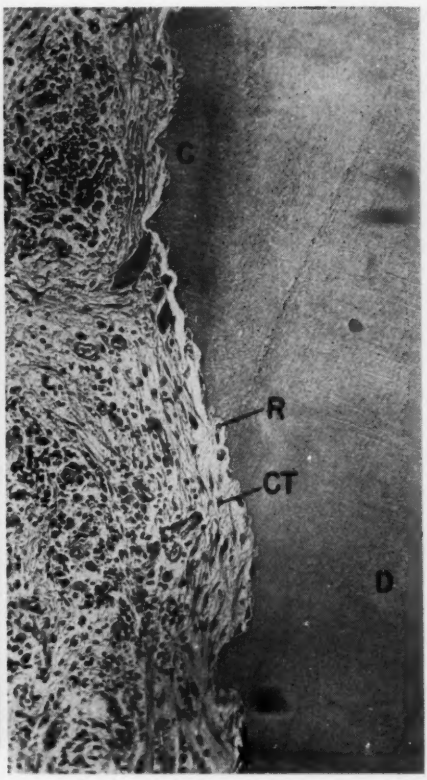

Fig. 8A. Inset of Fig. 8 magnified. Resorption bays (R) are noted with connective tissue cells parallel to the root.

therapy have manifested themselves by decreases in maximum biting force.[36]

The lack of correlation between histological and clinical results, the possibility of the probe plunging beyond pocket depth, and the complications introduced by denuding dentin all combine to make the pocket measurements of this study of doubtful value. The microscopic pictures furnish a more reliable index for interpretation.

The average gingival recession of slightly more than one-half millimeter is close to that recorded in previous studies.[33,34] It is probably due to the natural contraction of healing connective tissue which occurs whether or not union with the root is consummated.

Histological—Connective tissue healing took place against cementum in all cases except in the presence of infection (Fig. 3). Figures 6, 8, and 13 do not show healing to cementum. Chart 1 shows, however, that pre-operative and post-operative pockets existed in these cases. The cementum in question must have been pocket cementum, not part of the experimental surgical wound. In a previous experiment,[33] healing did not ensue across a naturally occurring crevice or pocket.

Healing occurred against cementum that was untouched (Fig. 7, 10, 11, 12, 6, 9),

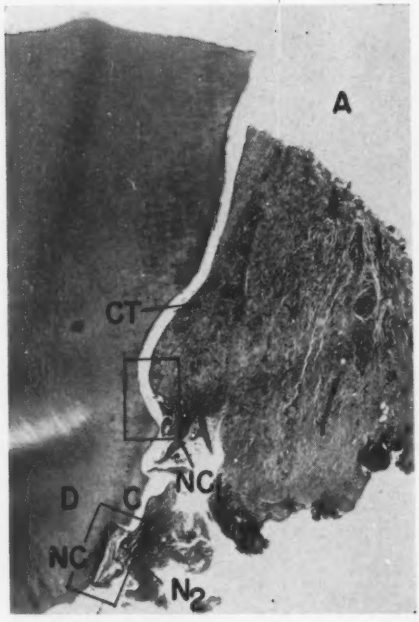

Fig. 9. Tooth with chronic pulp exposure and periapical radiolucency. New cementum (NC) appears in terminal nick (N_2). NC_1 is either new cementum torn loose or alveolar bone. A is artefact where epithelium was torn off.

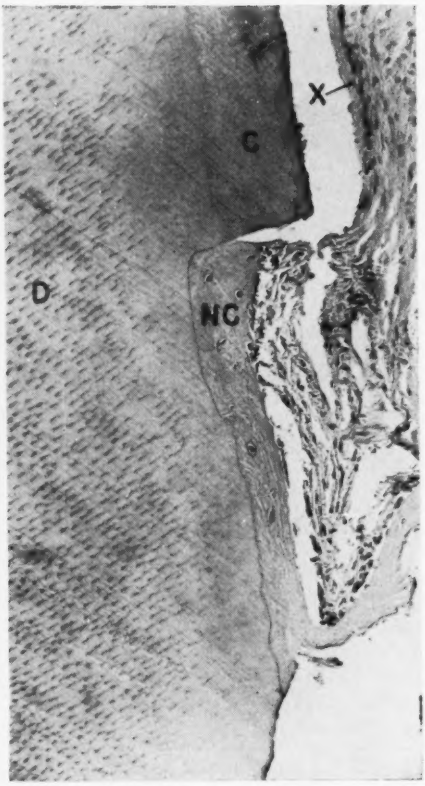

Fig. 9A. Inset of N_2 of Fig. 9 magnified. New cementum (NC) in terminal nick and cementoid (X) is deposited on old cementum (C).

curetted (Fig. 5, 13), or partially removed by instrument or bur (Fig. 8, 10, 13). Since it made no difference which cemental layer was exposed, it is evident that it is the intercellular cemental substance presenting the favorable environment for healing, rather than the outer vital or inner non-vital cementocytes.

Healing did not take place against the dentin of teeth with root canal fillings (Fig. 2, 5, 10, 11, 12) and tooth #8, and old carious exposure with a periapical area, fistula and unfilled root canal (Fig. 6). When the denuded dentin was in the coronal half of the wound (Fig. 2, 6, 6A, 10, 11, 13) no healing occurred, the epithelial lining growing past and attaching itself to the first surgically exposed cementum encountered (Fig. 6, 6A, 10, 12, 13). When dentin was isolated from the oral cavity by healing tissues (Fig. 5, 11, 12), the connective tissue merely filled the space. New cementum was observed (Fig. 5, 5A, 11), growing across the cut ends of the adjacent cementum, attempting to cover the dentin. This cementum creeps in from all borders of the artificial defect (Fig. 5C) quickly reaching the depth of the lateral ends of the nick which border the untouched cementum (Fig. 5B, 5C).

As previously reported,[33,34] the periodontal fibers generally formed themselves parallel to the root surface. Longer post-operative periods might have revealed a functional re-orientation.[33,39]

The results in this paper are in sharp contrast to the union resulting in the surgical wounds about vital teeth[33] and in the

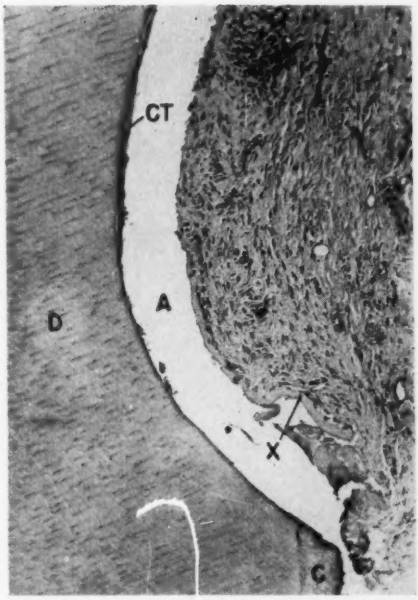

Fig. 9B. Inset of area coronal to N₂ of Fig. 9 magnified. Cementoid (X) is noted across artefact (A) from exposed dentin (D).

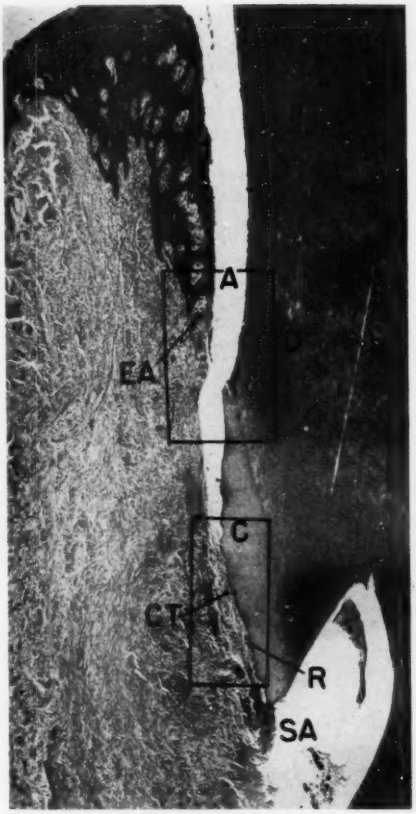

Fig. 10. Tooth with root canal filling with coronal cementum removed. Epithelial attachment (EA) is located opposite point where old cementum (C) remains. Surgical artefact (SA) removed the terminal nick.

surgical wounds apical to naturally occurring pockets about vital teeth[34] where the heaviest layers of new cellular cementum were observed depositing directly against the deepest dentinal deformities. They are, however, in complete agreement with observations made after apicoectomies[1, 10, 16, 18] and an experimental wound against a non-vital tooth in a dog.[40] These latter experiments presented the common situation of non-vital dentin bordering a healing periodontal area.

The results are also in agreement with those recorded on *vital* teeth of monkeys.[43] No explanation can be offered for this contradiction.

Two teeth with periapical areas and no root canal fillings, one with (Fig. 9) and one without a carious exposure (Fig. 4, 4A), both showed connective tissue union with dentin connected with the gingival crevice. The former showed profuse deposition of cementoid (Fig. 9B) against this dentin and new cellular cementum in the dentinal nick (Fig. 9A). These phenomena are characteristic of vital teeth and conform with Waerhaug's observations[45, 46] following a surgical wound about a human tooth with a gangrenous pulp but no root canal filling. It suggests that such teeth may have some residual vitality regardless of diagnostic tests.

Summarizing past and present evidence (as pictured in Fig. 14) the healing of periodontal connective tissues, following their surgical or accidental separation from the root, occurs in the following manner. Healing occurs directly with the dentin of vital teeth and cementum of vital or non-

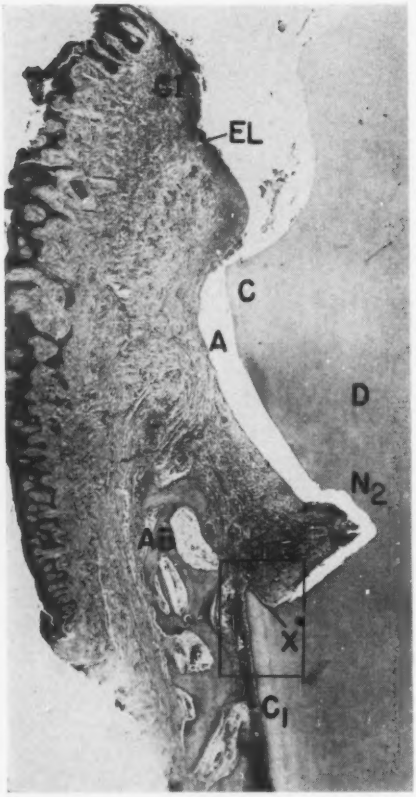

Fig. 11. Tooth with root canal filling with cementum removed from coronal end of wound. A pink staining material (X) is seen growing across old cementum (C) on to the dentin (D).

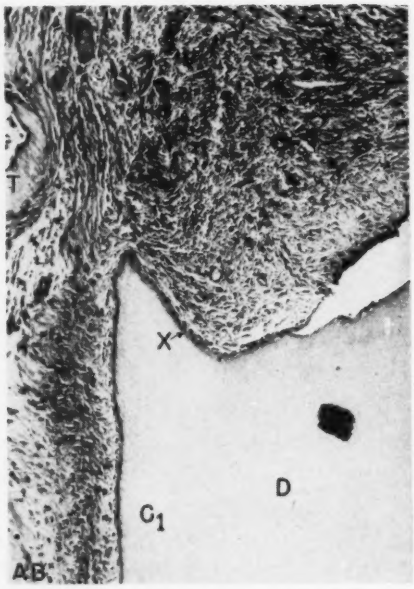

Fig. 11A. Inset of Fig. 11 magnified. Pink staining material (X) is growing from cementum (C) to dentin (D).

vital teeth and does not occur with dentin of non-vital teeth. When this non-vital dentin is connected with gingival crevice the epithelial lining grows past until the first surgically exposed cementum is encountered. When it is remote from the gingival crevice, a limited attempt is made to cover the area by growth of cementum from the undisturbed borders. When cementum is deposited it is thickest and most cellular in the deepest vital dentinal exposures, and thinnest and least cellular on the old cemental surfaces and toward the cervix of the tooth.

The reasons for lack of union with non-vital dentin must be related to changes that have taken place in that structure. These changes may involve a lack of fluid exchange, the absence of some inductive principle, or the medications used in root canal therapy.

Boedecker and Lefkowitz[11] have suggested that the pulp is an active regulator of fluid exchange in dentin. Thus, those teeth with gangrenous pulps that healed like vital teeth may have retained small areas with functioning odontoblasts. Another possibility is that the fluid exchange is a passive process independent of odontoblastic activity but is blocked by a root canal filling or the epithelial lining of a fistula (Fig. 6). Such a physiochemical explanation has been offered by experiments with radioactive isotopes, although the question has not yet been fully resolved.[2]

The likelihood of an inductive principle operating in or about a vital root to encourage cemental formation may be inferred from the work of La Croix[28] and

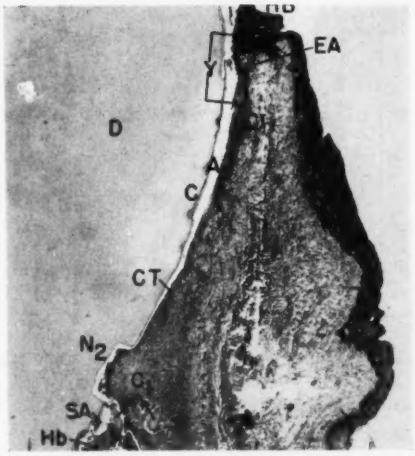

Fig. 12. Tooth with root canal filling. The epithelial attachment is located at the point (Y) where cementum (C) remained. Hemorrhage (Hb) from the biopsy procedure is seen near N_2.

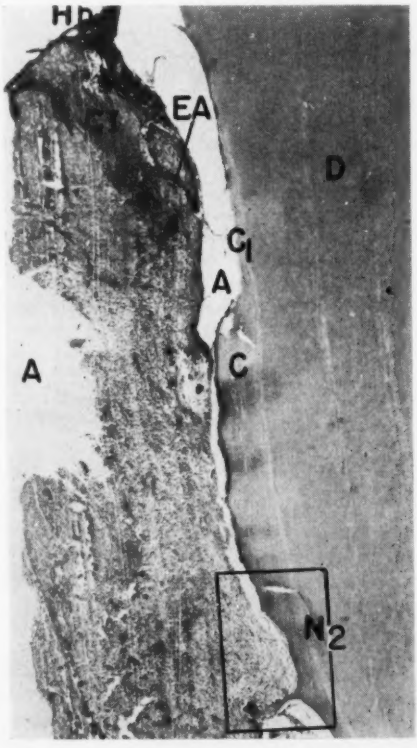

Fig. 13. Tooth with root canal filling where cementum was curetted. The epithelial attachment (EA) is located near the end of the remaining cementum (C_1). A cellular infiltration (CI) is noted adjacent to the epithelial attachment.

McLean and Urist[44] on bone. Their experiments showed that connective tissues may be induced to form bone under the influence of certain types of adjacent bone, cartilage or periosteum. Some support is lent to this thesis by the formation of a cellular calcified material noted around human dentin implanted in the femur of rabbits.[9]

The effects of root canal filling materials or sterilizing agents cannot be discounted. All the teeth were filled with gutta percha except #1 (Fig. 2) which contained a silver point. However, the sterilizing and cementing materials could not be identified.

Although the exact reason is still undetermined, the findings indicate that the metabolic status of the root side of a wound may prevent its successful healing with the periodontal connective tissues. Future work on naturally occurring periodontal pockets should determine whether failure in those instances is due to metabolic changes in the root, in the surrounding tissues, or in both. Such studies are now in progress.

SUMMARY

1. Healing occurred against both untouched and curetted cementum following surgical detachment of periodontal tissues about non-vital teeth.

2. Healing did not occur against the dentin of non-vital teeth. When the dentin was continuous with the gingival crevice, the epithelial lining grew past and connected with the cementum. When exposed dentin was isolated from the gingival crevice, cemental growth was noted starting from the bordering cementum.

3. The dentin of teeth with old carious exposures and periapical radiolucencies may show healing reactions similar to those of vital teeth.

The author wishes to express his appreciation to Drs. Barnet L. Levy and Frank E. Beube for their advice and cooperation, to Mrs. Florence Moore for obtaining patients, to Miss Mary Minasian for the preparation of the histological material, to Dr. Joseph Schroff for extending the courtesy of the Oral Surgery Department in obtaining the biopsy

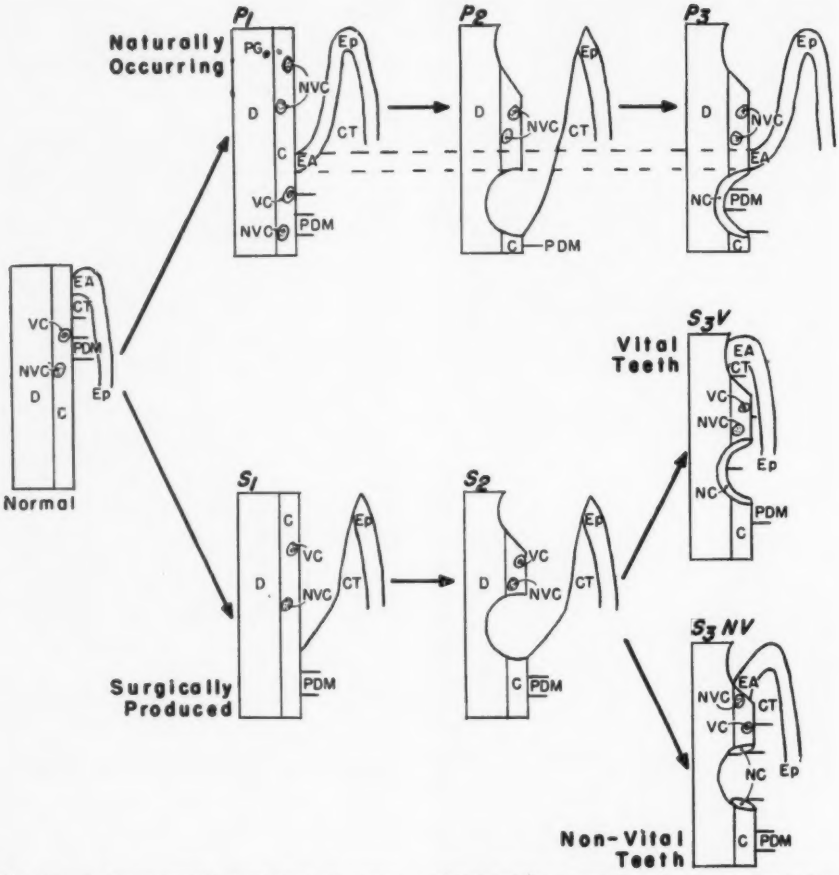

Fig. 14. A diagrammatic composite of experimental results to date. VC—Vital cementocytes. NVC—Non-vital cementocytes. CT—Connective tissue PDM—Periodontal membrane. EA—Epithelial attachment. EP—Epithelium. NC—New cementum. P.G.—"pathologic granules" of Bass.

The naturally occurring or pathological pocket (P_1) presents a root surface with non-vital cementocytes whose blood supply was cut off by the original pathology. The inner cells are non-vital, becoming this way as new cemental layers are added. Removing epithelium, cementum and some dentin produces P_2 which histologically is the same as S_2, which is surgically produced. P_3 shows healing occurring apical to the level of the original pocket with deposition of new cementum in the same manner as surgical pockets in vital teeth (S_3V). Coronal to the old pocket depth a new pocket forms whether the root surface be cementum or dentin.

When the surgical pocket is against a non-vital tooth with a root canal filling (S_3NV), the epithelial lining passes denuded dentin and stops at the cementum. Exposed dentin apical to this union does not show direct deposition of cementum as in vital teeth (P_3,S_3V) but shows cemental growth from the nick borders, attempting to cover the gap.

material through the personal efforts of Drs. Arnold Baumarsh and Julian Anderson and Miss Elise Boyd, and to Dr. Phil Silverstein for the drawing of Figure 5C.

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The Dental Department of the Beth Israel Hospital announces a postgraduate course in Periodontia to be given in the Fall of 1957.

A two-week seminar in Periodontia is scheduled for October 7 to October 18. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations, and clinics. The tuition fee is $350.00.

Co-Directors of the Course: Henry M. Goldman, D.M.D., Bernard Chaikin, D.M.D. For further information, write to: Director of Public Relations and Education, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

Tissue Reaction to Metal Wires in Healthy Gingival Pockets*

by JENS WAERHAUG, D.D.S., PH.D., *Oslo, Norway*

REPORTS on the reaction of the soft tissue to metal ligatures placed in healthy gingival pockets seem to be scarce. In an experiment on dog Morgenroth[4] placed a steel wire (whether or not it was stainless steel is not mentioned) slightly below the gingival margin of a tooth for two months. His conclusion was, that ligatures cause severe damage to the gingival margin. Apparently on the basis of the foregoing experiment Meyer[3] warned against severing the connection between enamel and epithelium, as the pockets, thus created, will persist.

In previous works,[7,9,10] it has been shown that there is no attachment, in the real sense of the word, between the tooth surface and the epithelial cuff (epithelial attachment), and that the bottom of the clinical pocket is to be found at the deepest point of the latter, not as previously stated[2] at its outer border. When no calculus or plaque is present, such a pocket is usually sterile.[10] It has also been demonstrated[8] that the gingiva, under favorable conditions, can adjust to the surface of an acrylic crown in the same way as a normal epithelial cuff adjusts to the enamel surface.

The reaction of bone and connective tissue to metal implants has been rather extensively studied, and all the base metals, except vitallium, seem to be more or less irritating;[6] for survey of the literature the reader is referred to Bernier et al.[1] and Venable et al.[6]

For the purpose of space maintenance in the deciduous denture Sannerud[5] introduced a simplified method based on the application of stainless steel wires around the teeth that were to be prevented from migrating. These steel wires were placed at the bottom of the pocket and thoroughly tightened so that they completely disappeared under the gingivae. Clinically it was found that the gingival structures healed readily around the stainless steel wires and no ill effects were observed in 972 cases.

During orthodontic treatment and fixation of teeth in periodontal cases ligatures are frequently placed below the gingival margin of pockets. The purpose of the present investigation is to find out whether a permanent damage will be the result or a more or less complete healing may be expected.

MATERIAL AND METHOD

The experiment was carried out on 21 teeth in 4 dogs and 2 teeth in one monkey. The animals were young and healthy, and there was no inflammation in the gingival margin as far as this could be determined clinically. Soft stainless steel ligatures (0.25 mm. thick) were applied around the teeth in such a way that they slid down to the bottom of the pockets when they were tightened. The twisted ends were then cut in a length of about 1 mm. and the wire was pressed down to the bottom in this area also. In most cases the ends disappeared completely under the gingival margin, in other cases they extended slightly above it. In a few cases ligatures of annealed iron and brass were used. The ligatures were left in the pockets for observation periods varying between 7 days and 206 days. As it was of particular interest to study the healing after the removal of the ligatures, these were in a number of cases removed some time before the end of the experiment. The animals were sacrificed by decapitation in pentobarbital sodium anesthesia. The jaws were taken out with great care and fixed in 10 per cent formalin for at least 2 days. They were decalcified in 5 per cent nitric acid, and cut into blocks suited for sectioning while kept in 70 per

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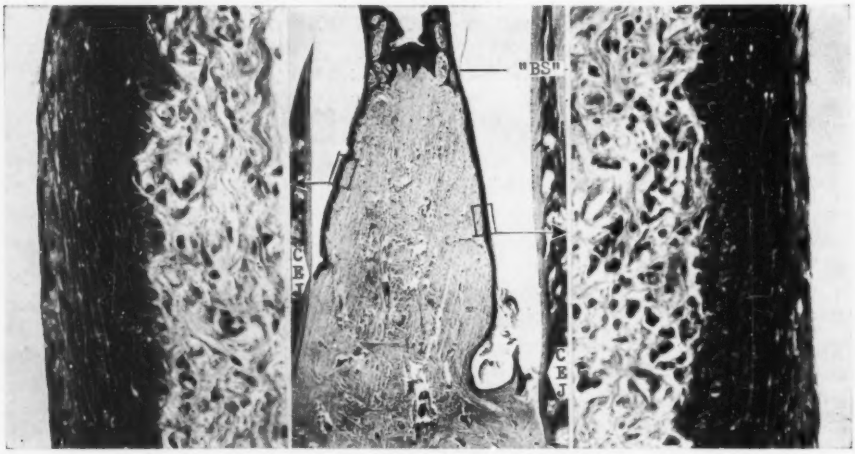

Fig. 1. Dog 56 LL 2, dist. Stainless steel ligature placed at bottom of clinical pocket 7 days before mors. Wire completely covered with apparently normal epithelium. Epithelial cuff (higher magnification to the right) re-adjusted to enamel and comparable to epithelial cuff on control tooth (left). "BS" indicates bottom of sulcus as situated according to the theory about the "epithelial attachment."

cent alcohol. These specimens were embedded in paraffin, and, the paraffin being thoroughly cooled down, the wires were cut off opposite the twisted ends and pulled out by these. In this way the tissue was prevented from being destroyed, which might have been the case if it had not been kept rigid by the paraffin. The specimens were then once more transferred to the paraffin oven in order that the spaces left by the wire be filled in. Some of the specimens were cut in a bucco-lingual direction and others, in a mesio-distal direction. The entire tooth was sectioned and approximately every tenth section was stained with hematoxylin-eosin.

OBSERVATIONS

The animals were examined regularly during the observations period. Where stainless steel wires had been used, the healing was apparently complete after one week or more, except for a small area corresponding to the twisted ends of the wire, where slight inflammatory signs could be observed. In cases where annealed iron and brass wires had been used, there seemed to be more inflammation. The examination of the sections* revealed a certain variation in the reaction, for which reason 7 photomicrographs have been selected to illustrate some typical conditions. The wire was seldom found exactly at the cemento-enamel junction as in Figure 1. In some cases it was found slightly above (Figs. 2 and 6), in other cases below the cemento-enamel junction, embedded in the periodontal fibers (Figs. 3 and 4). This is what could be expected as the cemento-enamel junction never runs like a straight line. Whether the wire was found within the pocket (Figs. 1, 2 and 6) or below it (Figs. 3 and 4), it was always covered by epithelium. The epithelial lining adjacent to the stainless steel wires and even the annealed iron wires showed little or no signs of irritation (Figs. 1-4). In Figure 4 corrosive elements on the surface of the iron wire have been retained in the section (see higher magnification to the right), and it is

*The tissue around the brass wires was marred by discoloration, apparently on account of some chemical reaction between the metal and the nitric acid. Therefore, these sections could not be judged with the same degree of certainty as when stainless steel and iron wires had been used. Besides, they were not suited for photographing.

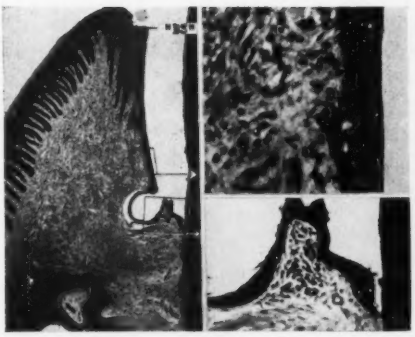

Fig. 2. Monkey, UR 6. Stainless steel wire placed at bottom of clinical pocket 23 days before mors. Apparently normal epithelial cuff adjusted to wire, and epithelial cuff between wire and gingival margin re-formed (higher magnification at the top to the right). Slight infiltration of round cells between wire and tooth (higher magnification at the bottom to the right).

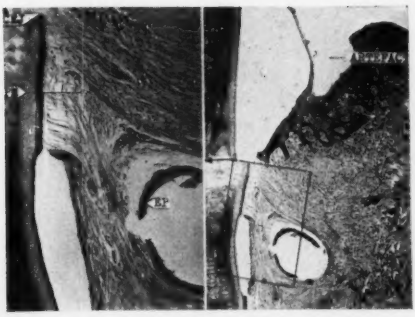

Fig. 3. Dog 56, LL 2, mes. Stainless steel wire placed at bottom of pocket and tightened 7 days before mors. Wire pressed into supraalveolar periodontal fibers. Periodontal fibers coronally to wire regenerated. Periodontal fibers between wire and tooth orientated parallelly to tooth surface. Attachment to cementum apparently lost, partially or completely. Wire covered with epithelium, EP. No inflammation around wire.

seen that normal epithelial cells are joining closely to these. In the connective tissue corresponding to the wire, little or no inflammation could be observed (Figs. 1-4), and if present it was usually found in areas where the wire had lain in contact with the tooth (Fig. 2 at the bottom to the right). In cases where the wire had penetrated into the periodontal fibers, these were usually regenerated up to the cemento-enamel junction (PM, Fig. 3 to the left). However, between the wire and the tooth the connective tissue fibers were usually orientated parallelly to the tooth surface. Both in cases where the wire lay within the pocket (Figs. 1 and 2) and in those where it lay below it (Figs. 3 and 4) a normal, or approximately normal, epithelial cuff had been re-formed in contact with the enamel.

In a few cases the ligatures were covered by a thin layer of bacterial plaque (Fig. 6 to the left). This was mainly the case when annealed iron wires had been used. In such cases it was also found that the bacterial plaque had grown along the tooth surface apically to the wires (P, Fig. 6 to the right). When bacterial plaque was present, degeneration of the epithelium, formation of rete pegs and severe inflammation in the underlying connective tissue were observed (Fig. 6).

In cases where the ligature had been removed some time before the animal was sacrificed, the healing was usually complete as in Figure 5. In this particular case wires had been placed around the 2 teeth and removed 71 days before the animal was sacrificed. The spaces left by the wires have been filled in, the epithelium, that most likely covered them, has disappeared, and a completely normal epithelial cuff has been re-formed. In this case no downgrowth of epithelium below the cemento-enamel junction could be found, although such a downgrowth was observed in some cases. (Table I and II). In a single case where the ligature had been removed, subgingival bacterial plaque was observed, Figure 7. As was the case around the wires where plaque had been formed, the epithelium was degenerated and thickened, and the connective tissue was inflamed in areas corresponding to the plaque. In the case of Figure 7 there was also an apical migration of the bottom of the pocket of 1.7 mm.

In Table 1 and II some of the main data concerning the experiment have been tabulated (see legend).

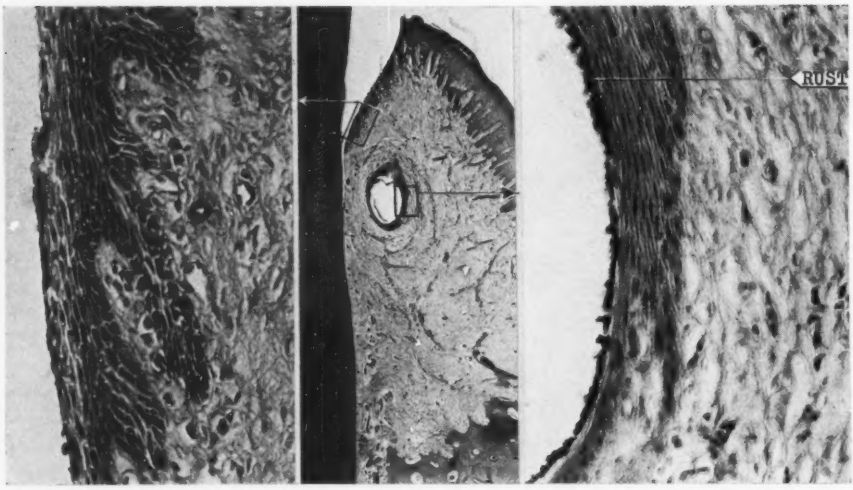

Fig. 4. Dog 54, LL 10, buc. Annealed iron wire placed in pocket and tightened 206 days before mors. Wire pressed into supraalveolar periodontal fibers. Apparently normal epithelial cuff joins the corrosive elements on surface of wire. No inflammation in underlying connective tissue. Epithelial cuff re-adjusted between cemento-enamel junction and gingival margin (left). Note, in other areas wire covered with bacterial plaque as in Figure 6.

DISCUSSION

As revealed from this experimental series, it is obvious that the stainless steel wires are well tolerated by the soft tissue. It is apparent that ligatures placed in healthy pockets are rapidly covered by epithelium even when pressed into the periodontal fibers (Figs. 3 and 4). It is, consequently, the reaction of the epithelium to the wires which is studied in these series. Stainless steel, iron and brass in contrast to vitallium, are not considered[1, 6] to be non-irritating to the tissue. When these metals have been so well tolerated in this experiment, it may be caused by the fact that they have been overgrown with epithelium. In earlier experiments[6] where the reaction to metals has been studied, these were covered by connective tissue. In most cases the conditions at the junction between epithelium and wire could not be studied with too great a degree of certainty, as the wire had to be pulled out. The cells may in this way have been damaged, and some of them may even have been pulled out with the wire. However, in Figure 4 the corrosive elements on the surface of the annealed iron wire have been retained in the section and it can be seen that normal epithelial cells join these elements in the same way as the "epithelial attachment" joins the tooth surface.

The great ability of healing of gingival tissues experienced in earlier experiments,[7, 8, 9, 10] was observed here as well, and it is quite remarkable that the healing is nearly complete after 7 days (Fig. 1 and 3). A round, rather well polished wire, will furnish bacteria little or no chance of retension, and after such a wire has been firmly fastened to the tooth, either within the pocket or between the periodontal fibers, the defense mechanism of the body has no difficulty in destroying the bacteria that were inserted with the wire. If the metal happens to be inert or very little irritating, the epithelium will adhere directly to the ligature.

During the insertion of the wire the epithelial cuff had to be loosened from the tooth surface. Therefore, it is remarkable

TABLE 1

Data concerning observation period maximum pocket deepening, plaque formation and inflammation (− lack of inflammation, + slight inflammation, ++ conspicuous inflammation) around wires of stainless steel, brass and annealed iron placed below gingival margin in 4 dogs and 1 monkey. Pocket deepening was usually limited to smaller areas, but in this table only maximum values are given. Subgingival plaque, if present, was not usually found in all areas, but if present at all, it is tabulated here.

Animal	*Tooth*	*Obs. per. (days)*	*Pocket deepen. in mm.*	*Plaque on wire*	*Inflam. around wire.*	
D 54	LL 1 D	193	0.0	−	−	
"	" M	"	0.0	+	+	
D 54	LR 1 M	193	0.0	−	−	
"	" D	"	0.4	−	−	
D 55	LL10 L	71	0.0	−	−	
D 56	UR 2 B	13	0.0	−	−	
"	" L	"	0.0	−	−	
D 56	UL 1 B	13	0.0	−	−	
"	" L	"	0.0	−	−+	
D 56	UL 3 B	7	0.0	−	+	Stainless
"	" L	"	0.0	−	+	steel.
D 56	LL 2 M	7	0.0	−	−	
"	" D	"	0.0	−	−	
D 57	LL 1 M	95	0.1	−	−	
"	" D	"	0.4	−	−	
D 57	LL 2 B	95	0.0	−	−	
"	" L	"	0.0	−	−	
D 57	LL 3 M	95	0.1	−	−	
"	" D	"	0.2	−	−	
Monk.	UR 6 B	23	0.8	−	+	
"	" L	"	0.0	−	+	
Monk.	UL 6 B	23	0.3	−	−	
"	" L	"	0.2	−	−	
D 54	LL11 B	206	0.9	+	++	
"	" L	"	1.0	+	++	
D 54	LL10 B	206	0.2	+	++	Iron
"	" L	"	0.3	+	++	
D 55	LR 5 B	87	0.0	−	+−	
"	" L	"	0.0	−	+−	
D 55	LL 5 B	87	0.6	−	+	
"	" L	"	0.0	−	+	
D 57	LR 3 B	131	0.0	−	+	Brass
"	" L	"	0.0	−	+	
D 57	UR 2 M	131	0.0	−	+	
"	" D	"	0.0	−	+	

TABLE 2

Data concerning observation period, pocket deepening and plaque formation around 5 teeth in 2 dogs where the wires had been removed some time before the animals were sacrificed. + indicates number of days the wire was in place and − indicates number of days since wire was removed. Healing was complete in all cases but D 54, UR 4, ling. (Fig. 7) where subgingival plaque remained below gingival margin.

Animal	*Tooth*	*Obs. per. (days)*	*Pocket deepen. in mm.*	*Presence of plaque in pocket*
D 54	UR 4 B	+135–71	0.6	−
"	" L	" "	1.7	+
D 54	LR 4 B	+135–71	0.0	−
"	" L	" "	0.0	−
D 54	UR 2 D	+135–71	0.0	−
"	" M	" "	0.0	−
D 54	UR 1 D	+135–71	0.0	−
"	" M	" "	0.0	−
D 57	UR 2 M	+ 45–56	0.0	−
"	" D	" "	0.0	−

that a new and normal epithelial cuff has readjusted to the tooth between the wire and the gingival margin, and that the enamel-epithelial junction appears similar as on untreated teeth (Figs. 1 and 2). This experiment demonstrates also that the bottom of the clinical pocket, as experienced during dental treatment, is to be found at the *deepest* line of the "epithelial attachment." According to the theory about the "epithelial attachment" the bottom of the sulcus in Figures 1 and 2, should be found at "BS." The splits above "BS" in these illustrations are obvious artefacts and have nothing to do with the bottom of the pocket as observed clinically. Therefore, pictures like these help to clarify the correlation between the clinical and the histological concept of the pocket. The fear of severing the connection between the epithelium and the enamel[3] seems, therefore, to be unwarranted.

As demonstrated in Figure 6, the wire was in some cases covered by bacterial plaque; and as this plaque was found only on one of the stainless steel wires while it was observed on two of the three annealed iron wires (Table I), there is reason to believe that the rough surface of the rust facilitates the retention of bacteria. Where bacterial plaque was retained, the inflammatory reaction was apparent (Fig. 6, Table I), and of the same character as when the plaque was retained on the tooth surface. This bacterial inflammation caused

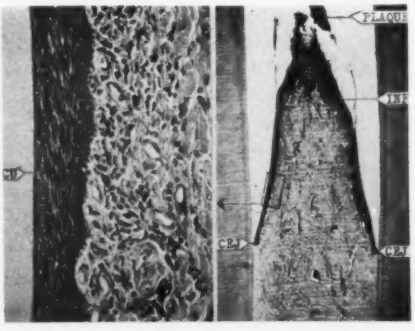

Fig. 5. Dog 54, UR 2, mes. and UR 1 dist. Stainless steel ligatures placed in pockets for 135 days and removed 71 days before mors. Complete healing with re-formation of normal epithelial cuffs (left). Inflammation in upper part of papilla caused by bacterial plaque in contact with gingiva.

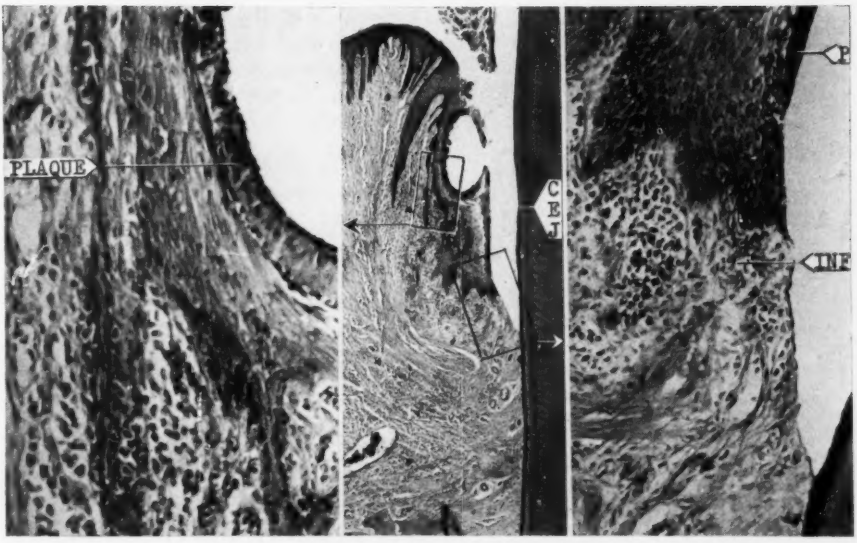

Fig. 6. Dog 54, LL 10, ling. Annealed iron wire placed in pocket 206 days before mors. Wire covered with bacterial plaque (left). Plaque, P (right), reaching close to deepest point of epithelial cuff. Epithelium thickened and degenerated correspondingly by round cells. Downgrowth of epithelium below cemento-enamel junction, CEJ, 0.3 mm.

a break-down of the periodontal fibers and facilitated downgrowth of epithelium below the cemento-enamel junction.

If a wire covered with plaque is removed, the plaque on the tooth surface below the gingival margin may be retained there; and that is what has happened in the case of Figure 7. In a previous work[10] it has been stressed that polymorphonuclear leukocytes are not able to destroy bacteria when these are entangled in the filamentous stroma of Actinomyces and Leptothrix. The main damage caused by a wire placed below the gingival margin seems to be that bacterial plaque may grow along it and thus be retained on the tooth surface. There is reason to believe that plaque is most easily retained on the twisted ends of the wire which extend out of the pocket. Therefore, this constitutes the weak point.

In earlier experiments,[7,9,10] it has been demonstrated that complete healing may be attained after subgingival calculus is removed, i.e. a normal epithelial cuff may be re-established. When space maintainers of the type described by Sannerud,[5] or other similar ligatures are used on permanent teeth, a thorough scaling should be undertaken after removal of the wires and, if possible, the tooth should be polished below the gingival margin with a rotating rubber cup filled with pumice. The cleansing should be particularly thorough in the area where the twisted ends have been placed. In that way a result as demonstrated in Figure 7 can be prevented.

In the majority of cases the healing after removal of the wires was complete as demonstrated in Figure 5. This observation together with Sannerud's clinical experience of the gingival condition being undisturbed by the use of his space maintainer, justifies the conclusion that no serious damage will be caused if the precaution mentioned above is taken.

In Table I it may be seen that a certain amount of pocket deepening had occurred in limited areas adjacent to some of the teeth. This downgrowth of epithelium was usually observed where the wire was

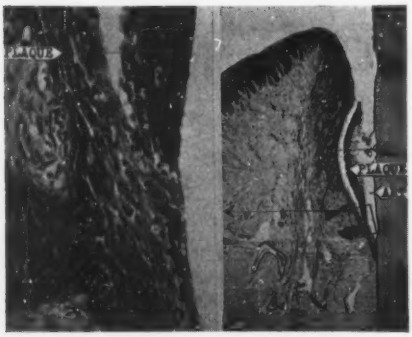

Fig. 7. Dog 54, UR 4, ling. Stainless steel wire placed in pocket for 135 days and removed 71 days before mors. Subgingival bacterial plaque extending close to the deepest point of epithelial cuff. In area corresponding to plaque, thickened and degenerated epithelium (right). Infiltration of round cells in underlying connective tissue. Bottom of pocket 1.7 mm. below cemento-enamel junction, CEJ.

situated just below the cemento-enamel junction. In Figure 3 it may be seen that Sharpey's fibers between the wire and the tooth apparently have lost their firm connection with the tooth. If the wire had been placed just below the cemento-enamel junction, the possibility of a downgrowth of epithelium would have been present. The tendency of primary healing of the periodontal membrane is also demonstrated in Figure 3, as there is no sign of these fibers having been severed one week earlier.

This material does not allow any definite conclusions with regard to the difference in reaction to the three materials used, but it seems to be justified to presume that stainless steel is the material of choice for ligatures which have to be placed below the gingival margin. Even if stainless steel is not considered to be altogether inert to living tissue, the irritation, if any, is so slight that it will hardly cause any mentionable damage when used over a limited period of time.

CONCLUSIONS

1. Stainless steel wires that are placed in healthy gingival pockets for orthodontic purposes are usually well tolerated by the soft tissue.

2. During a week stainless steel wires may be completely covered by epithelium, which apparently adheres to them in the same way as a normal epithelial cuff adheres to the tooth surface.

3. The epithelial cuff between the steel wire and the gingival margin will usually regenerate and approximately normal conditions will be re-established in the pocket.

4. When the ligatures are pressed into the periodontal membrane below the cemento-enamel junction the connective tissue fibers coronally to the wires will usually regenerate in a short time, although a limited downgrowth of epithelium may be expected in areas where the wire lies just below the cemento-enamel junction.

5. Iron wires, although comparatively well tolerated by the tissue, seem to facilitate retension of bacterial plaque and should hardly be used.

6. Bacterial plaque which has grown along the wire may, under favorable conditions, be retained on the tooth surface below the gingival margin, and remain there, after the wire is removed.

7. When removing orthodontic ligatures from permanent teeth, the tooth surface should be thoroughly cleansed so that any subgingival bacterial plaque is completely removed.

8. When the precaution mentioned under point 7 is taken, a complete healing with re-establishment of a normal sterile pocket may be expected after the removal of the wire.

9. The application of stainless steel wires in healthy gingival pockets in young individuals may be considered a justified method.

SUMMARY

Around 21 teeth in 4 dogs and 2 teeth in 1 monkey ligatures of stainless steel, iron and brass were placed in healthy gingival pockets. Histological sections after 7 days to 206 days showed that the ligatures

were well tolerated by the tissue, stainless steel being apparently the least irritating. The ligatures were in some cases found within the pocket and in other cases embedded in the supraalveolar periodontal fibers. In both cases, after a comparatively short period of time, they were completely covered by epithelium, which apparently joined them in the same way as an epithelial cuff joins the tooth surface. The epithelial cuff between the wire and the gingival margin was in most cases regenerated and approximately normal conditions were reestablished. In a few cases the wires seemed to facilitate retension of a bacterial plaque. A slight localized pocket deepening could be observed in some cases.

In five cases where the wires had been removed some time before the end of the experiment, complete healing was observed in all cases except one, in which subgingival bacterial plaque persisted.

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POSTSCRIPT*

The present findings should be viewed on the background of recent article by Orban, Bhatia, Kollar, and Wentz, entitled: The Epithelial Attachment (Attached Epithelial Cuff), J. Periodont. 27:167, 1956. Referring to one of my previous works[7] they contend "that by some mistake, Waerhaug's sections were made from a tooth or an area of a tooth that had not been subjected to the experimental procedure" described to the text. It is to be hoped that similar comments will not be repeated concerning the experiments presented here.

It is hard to understand how Orban et al. can make a generalized statement that the bottom of the sulcus, in cases similar to their Figures 1 and 2, is located at "BS" when it is clearly demonstrated that the ligatures easily slid down to the area of the cemento-enamel junction. Of course, a steel blade or a Mylar strip can be stopped at "BS" if sufficiently little force is used, but that must be considerably less than the forces in clinical procedures such as probing and scaling.

The concept of the "epithelial attachment" as a rigid system, which, once broken, is damaged forever, is also disproved in this series. The epithelial cuff is a dynamic system which will adapt itself to any surface that is non-irritating. To speak of an epithelial attachment to corrosive elements, (Fig. 4) "similar to that existing between a muscle or tendon to bone" would hardly make sense.

The present author has repeatedly mentioned[7] that one or two layers of cells usually are retained on the tooth surface when the epithelial cuff is separated from it. However, these cells are so loosely attached that they can be sprayed away with a jet of water, which demonstrates that the attachment is extremely weak. Therefore, when Orban et al., found the split within the epithelium when steel bands were in-

*This postscript was added after the galley proofs of this article were returned to the editor.

serted, this does not prove that the epithelial cells are attached to the tooth in a way similar to a muscle attachment. Furthermore, it must not be forgotten that the surface layers of the epithelium degenerate and that the intercellular attachment decreases. Therefore, the attachment between the cells in the outer layers of the epithelial cuff cannot be expected to be strong, of any.

The epithelial cuff in dogs is approximately 0.025 mm. thick. When such an extremely thin layer of cells is separated from the tooth surface with an instrument which is at least twice as thick (0.05), it is not to be expected that this will always follow the tooth surface.

In the description of their method Orban et al. do not specify the amount of force used when steel blades or Mylar strips were inserted into the pockets. At the same time they repeatedly emphasize that "the fibers surrounding the tooth, guarantee the firmness of the junction between soft and hard tissues." The question then arises: was the force they used greater than that required to overcome the friction caused by the connective tissue pressing the epithelium against the tooth, together with a certain amount of adhesion of epithelial cells to the latter?

The Value of Step-Serial Sectioning in the Morphologic Study of Human Periodontal Disease

by HAROLD R. STANLEY, JR.,* *Bethesda, Md.*

IN the study of periodontal disease by histological methods, the relationships between various components of the periodontium, either normal or pathologic, are evaluated, in part, on the basis of distances between certain landmarks which can be observed in the sections. Two recent investigations have suggested that these relationships can be more precisely described and compared metrically than by visual estimate.[1,2] Certain features in sections are well enough delimited so that the distances between them can be measured, and the data thus derived can be subjected to statistical analysis. To date, the sections have been selected arbitrarily, according to the custom in most histological studies, and presumed to be representative of the blocks of tissue from which they have been cut. Because of the uncertainty connected with the assumption that a selected section is a representative sample of the region sectioned, the present investigation was undertaken to determine the variation in measured distances that might be revealed in serial sections.

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MATERIAL

Human specimens, three obtained surgically and one at autopsy, were used in this study. One specimen was an extracted tooth with its buccal bony plate and soft tissues intact; the others consisted of blocks of two teeth and their intervening periodontal tissues.

The specimens were fixed in 10% formalin, decalcified in 5% formic acid, washed, dehydrated, imbedded in paraffin, and serially sectioned at six microns. The sections were stained with hematoxylin and eosin. All sections of a block obviously can not be used since in cutting either from a buccal or lingual direction, the curvature

and convexities of the teeth and soft tissues are such that many sections do not present an intact, composite picture of a tooth with its periodontium. Consequently, these sections are eliminated. If more than an occasional section of a successive series were lost due to technical difficulties, that area, possibly involving several hundred microns of tissue would of necessity be eliminated. Many more blocks were serially sectioned than included in this study but only four provided successive sections numerically adequate for evaluation.

To clarify the location of the areas examined the term "surface" is used to include those soft tissues, between the gingival crest and the alveolar crest, which approximate or attach to the root surface contiguous with the clinically designated crown surface. For example, if the periodontal tissues on the mesial root of a tooth were studied, the area would merely be designated as the mesial surface. Four interproximal and two labial surfaces were appraised. These surfaces represent typical periodontal lesions of varying degrees of involvement. The history of the cases and description of the specimens are as follows:
Case I (540013)

Surface #1 (mesial of right mandibular second molar), serial sections represent approximately 2100 microns of the specimen.

This surgical specimen came from a 69 year old, white male with squamous cell carcinoma of the floor of the mouth, extending into the right mandible and submaxillary gland. The tumor did not directly involve the periodontal tissues. Microscopically, the specimen exhibited advanced, severe periodontitis simplex. The lining epithelium, approximating large nodules of calculus, presented acute, inflammatory lesions. The interdental papilla was heavily infiltrated with chronic inflammatory cells. The alveolar crest exhibited active bone resorption.
Case II (540055)

Surface #2 (distal of right mandibular lateral incisor), serial sections represent approximately 1100 microns of the specimen; Surface #3 (mesial of right mandibular cuspid), serial sections represent approximately 1100 microns of the specimen; Surface #4 (labial of right mandibular cuspid), serial sections represent approximately 500 microns of the specimen.

This surgical specimen came from a 55 year old, negro male, also a victim of squamous cell carcinoma of the floor of the mouth, with involvement of the tongue, right half of the mandible and right submaxillary lymph nodes. The tumor did not directly involve the periodontal tissues. Microscopically, the distal and mesial surfaces presented a picture of chronic periodontitis simplex but not as severe or advanced as surface #1. The mesial surface, but not the distal, approximated calculus. The alveolar crest showed no active resorption. The labial surface, with a crevice of approximately normal size and devoid of calculus, exhibited only mild chronic gingivitis. The labial alveolar bone was dense.
Case III (550088)

Surface #5 (distal of left, mandibular first bicuspid), serial sections represent approximately 900 microns of the specimen.

This autopsy specimen came from a 47 year old, white female with adenocarcinoma of the breast. Metastases were found in the liver, lungs, brain, bones and multiple lymph nodes. Microscopically, a very slight gingivitis existed. No calculus was present.
Case IV (550082)

Surface #6 (labial of right maxillary cuspid, divided into blocks i and ii), serial sections from block i represent approximately 1000 microns, serial sections from block ii represent approximately 1100 microns of the specimen.

This surgical specimen came from a 32 year old, white male who was participating in a periodontal study in which pockets, created by surgery, were held open by the placement of steel bands. No other treatment followed. Microscopically, chronic periodontitis simplex of a slight degree was found. No calculus was present.

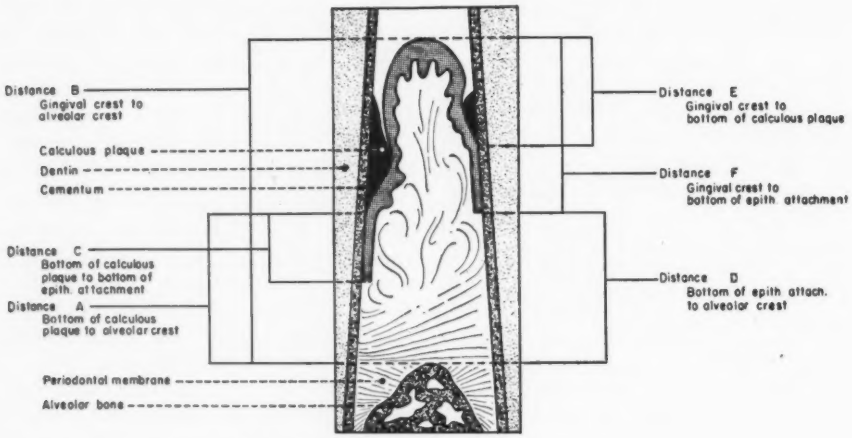

Fig. 1. Diagrammatic sketch of an interdental papilla.

METHOD

Six specific distances were designated for measurement at a magnification of x100. A stage micrometer was employed. It was felt that differences in these distances would reflect changes in the anatomic components as successive sections of a block were studied. These distances were (Fig. 1): (A) bottom of calculous plaque to alveolar crest, (B) gingival crest to alveolar crest, (C) bottom of calculous plaque to bottom of epithelial attachment, (D) bottom of epithelial attachment to alveolar crest, (E) gingival crest to bottom of calculous plaque, and (F) gingival crest to bottom of epithelial attachment (for those surfaces having no calculous plaque).

The rationale for selecting four of these distances was derived from our previous experience.[2] By comparing coefficients of variation, it was formerly demonstrated that distance A was the most constant and distances B, C and D followed in a decreasing order of constancy.

No attempt was made to differentiate calcified and uncalcified calculus. All measurements involving the bottom level of the calculus were taken at the lowest point of the accumulated debris directly beneath the calcified structure. Therefore, the landmark is referred to as the "bottom of the calculous plaque." Measurements utilizing the uppermost point of the epithelial attachment were not employed because of the difficulties in appraising the exact uppermost point of attachment.

RESULTS

Initially the distances were measured in every section, but it soon became obvious that the study of adjacent sections, or every second, third, or fourth section was redundant because the changes, if any, in the measurements and histologic characteristics were too minute to be significant. Therefore, every fifth section was selected as the minimal interval for study.

After the measurements were made on every fifth section (approximately 33 sections/mm. of tissue), the difference in value between two successive fifth-sections was found rarely to exceed 0.2 mm. Therefore, a preliminary statistical study was run to determine the greatest possible interval between sections that would yield data comparable to every fifth section. Intervals of 10 and 15 still appeared too close but intervals of 25 and 30 were definitely too great. Consequently, statistical tests† (mean, standard deviation, standard error of the mean and analysis of variance) were employed to determine whether or not every twentieth section (approximately 8-10 sections per mm.) would be satisfactory. Series of 20th sections were selected to permit the use of every fifth section. That is, one series began with section #5, others with section #10, #15 or #20 so that four series of twentieth sections could be compared. The statistical analysis of the

TABLE I

Results of Measurements of Most Variable Distance on Each Surface
(all measurements in millimeters)

Surface	Distance	Total Number of 5th-Sections Examined	Means of the 5th-Section Series	Means of Four Twentieth-Section Series within each 5th-Section Series				F Value, Variance Between Series (a-d)
				a	b	c	d	
1	D	58	1.07± .04	1.10± .07	1.06± .07	1.08± .07	1.06± .07	0.073
2	D	34	0.68± .02	0.68± .02	0.66± .04	0.70± .02	0.68± .04	0.203
3	E	34	1.03± .03	0.99± .08	1.03± .02	1.02± .06	1.08± .05	0.366
4	F	17	1.16± .03	1.16± .02	1.23± .03	1.13± .05	1.15± .03	1.660
5	D	30	0.96± .03	0.96± .08	0.98± .15	0.93± .06	0.97± .05	0.084
6	D (block i)	33	1.02± .07	1.00± .14	1.04± .14	1.01± .16	1.05± .14	0.026
	D (block ii)	34	1.14± .03	1.18± .07	1.13± .08	1.09± .07	1.14± .07	0.254

measurements of the most variable distance on each surface is included in Table I (all measurements are in millimeters).

None of the mean measurements obtained from the series of 20th sections differed significantly from the means obtained from the measurement of every fifth section. With the mean rounded to a single decimal place, 20th section means were identical with fifth-section means in 24 instances, less by 0.1 mm. in two instances and greater by 0.1 mm. in two instances. Analyses of variance indicated that the differences in distribution between the four possible 20th section series in any one surface were negligible (Table I). In general, an F value of about 2.8 or higher is required to indicate a valid difference at the P= .05 level, and an F value of about 4.3 or higher is required to indicate a valid difference at the P= .01 level with data of this nature.

DISCUSSION

The data indicated that essentially the same results were obtained from the measurement of every 20th section as from every fifth section. The procedure requires, however, that the number of sections examined be sufficiently large to guarantee a valid estimate of the distribution, that is, provide a representative sampling. In the specimens of the size dealt with in this study, representative sampling seemed to be adequately described by eight to ten sections per millimeter of tissue.

Distances A, B, and C were so consistently constant on each surface that they were not included in Table I. With these distances the same results could be obtained with even a greater interval than every 20th section; but, in order to include the aberrations of the more variable distances, every 20th section must be appraised.

It is noteworthy that distance D (bottom of the epithelial attachment to the alveolar crest) is recorded as the most variable distance on four of six surfaces in Table I. Distances A, B, and C, in no instance variable enough to be listed in Table I, appear to be reliable aids for the morphologic comparison of periodontal lesions.

The severity of the periodontal lesions did not affect the value of the step-serial procedure. The disease process appears sufficiently uniform in a three-dimensional study, so that this procedure should be applicable to other "typical" human periodontal lesions.

SUMMARY

Because of the uncertainty that exists in accepting a single histologic section as representative of a three dimensional human periodontal lesion, this study was undertaken to ascertain by serial sectioning the degree of variation of certain measured

distances limited by anatomic and pathologic landmarks within the periodontium.

The material consisted of four human specimens, three obtained surgically and one at autopsy. Four interproximal and two labial surfaces, representing typical periodontal lesions, were appraised.

Six specific distances were designated for measurement at a magnification of x100. It was felt that differences in these distances would reflect changes in the relationships of these components as successive sections of a block were studied.

The data indicated that the amount of change was of the order of magnitude that essentially the same results were obtained by the measurement of every 20th section as every successive section or every fifth section. However, the procedure requires that the number of sections examined be sufficiently large to guarantee a valid estimate of the distribution, that is, provide a representative sampling. In this material, distribution appeared adequately described by 8 to 10 sections per millimeter of tissue.

The constancy of distance A (bottom of calculous plaque to alveolar crest), distance B (gingival crest to alveolar crest) and distance C (bottom of calculous plaque to bottom of epithelial attachment) further establishes them as reliable aids for the morphologic comparison of periodontal lesions.

The author is indebted to Albert D. Russell, Chief of the Epidemiology and Biometrics Section of the National Institute of Dental Research for his assistance and guidance in the development of the statistical analysis.

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Announcement

UNIVERSITY OF PENNSYLVANIA SCHOOL OF DENTISTRY

Applications are requested for a Fellow in Oral Pathology supported by the American Cancer Society, tenable at the Department of Pathology, School of Dentistry, University of Pennsylvania, for a period of one year beginning 1 August 1957.

A stipend of $3600 accompanies the fellowship.

Partial board credit granted (6 months) by American Board of Oral Surgery. Under proper conditions, credits may be awarded toward an advanced degree.

Each candidate should submit a curriculum vitae and letters of reference. Reply to Dr. C. E. Wilde, Jr., Department of Pathology, School of Dentistry, University of Pennsylvania.